PULMONARY FIBROSIS: WITH SPECIAL REFERENCE TO ITS OCCURRENCE IN STONE-MASONS: A CLINICAL AND THERAPEUTIC STUDY.

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The Term Pulmonary Fibrosis; Its Signification, With Some Account of Other Synonymous Terms.

Pulmonary Fibrosis may be defined as a condition of the lung in which there is a hyperplasia of fibrous tissue which tends to obliterate the air cells. This fibrous tissue may be formed in any part of the lung to a greater or less extent, and it always shews a marked tendency to spread from one portion of the lung to another until more or less of the respiratory organ is thrown out of action.

Various terms have been applied to this pathological change which takes place under quite a variety of circumstances. It may be advisable to look briefly at each of these alternative names in turn, One of the earliest terms given to this disease was that of Cirrhosis of the Lung which was applied to it by Corrigan of Dublin who regarded the change as very much akin to that which occurs in hepatic cirrhosis. Charcot and Wilson Fox used the term Chronic Pneumonia, believing it to be really a chronic inflammatory condition of the pulmonary tissue. Interstitial Pneumonia was the name given to it by Rotitansky among others,

who regarded the condition as actually occurring outside of and around the alveoli. The word "chronic" is sometimes prefixed to this nomenclature to indicate the more essential character of the process. Fibroid Degeneration of the Lung was the appellation first suggested by Handfield-Jones who looked upon the change as due to a constitutional tendency to the formation of fibroid tissue. More recently the late Sir Andrew Clark introduced the term Fibroid Phthisis which is, in my opinion, a most unfortunate one. The reasons for this difference of opinion will be referred to later, but meantime I may state that Clark's use of the word phthisis must be objected to on the ground that it suggests a tuberculous origin for all cases of pulmonary fibrosis, which is certainly by no means the case.

Melanosis, a term used by Bayle, is probably synonymous with pulmonary fibrosis. At any rate this word occurs in a work published by this French writer in the beginning of the last century who, however, seems to regard this disease as a complication of pulmonary tuberculosis, while in some of his cases bronchiectatic dilatations were present as well. His patients, however, were practically all over 60 years of age. Stokes in his work entitled "An Introduction to the Use of the Stethoscope", (published in 1825) refers to the phrase

"grey induration of the lung," which was originally employed by Andral in speaking of chronic pneumonia. The "ramollissement gris" of Andral is practically pulmonary fibrosis. "At other times," Stokes remarks, "the lung tissue is found to be of a pale red colour." Stokes also incidentally mentions a case of Bayle's which was mistaken for pulmonary tuberculosis but which was in reality one of "well marked chronic pneumonia." Later Addison used the term Grey Induration when writing of pulmonary fibrosis, and maintained that many of these cases occurred apart altogether from tuberculous infection, and that when tuberculous changes were met with in association with pulmonary induration they generally occurred as secondary complications.

We shall now consider the term Fibroid Phthisis adopted by Sir Andrew Clark in speaking of this disease. He maintained that there were three different degrees of fibroid disease of the lung:- (1) simple fibroid lung, (2) fibroid lung associated with bronchiectatic dilatations, and (3) fibroid lung with cavity formation to which he more especially applied the name Fibroid phthisis. There can be no doubt that this classification has come to be largely adopted by physicians both in this country and abroad, and

in some respects it is a convenient one. Still, at the same time, the phrase fibroid phthisis is very misleading. In defining the word "phthisis" Clark maintained that it "is that assemblage and progression of symptoms associated with and dependent upon the ulcerative or suppurative destruction of more or less circumscribed non-malignant deposits in the lung." By using the qualifying word "fibroid" he brought forward the particular variety of phthisis in which there is fibroid induration of the lung associated with cavities. He further pointed out that many of these cases have no tuberculous origin whatever; and when tubercle bacilli occur in the sputum then he would modify the nomenclature and classify the disease as tuberculo-fibroid phthisis.

In the first place the use of the word phthisis is distinctly objectionable. This name is simply the English rendering of the original Greek word φθίσις which was used in two senses. It signified either a wasting away or a decay, or was more specifically applied to persons with the meaning of atrophy or emaciation. It was used in the latter interpretation by Hippocrates, who also employed it in a more special sense as indicating decline or consumption equivalent to the Latin word "tabes". Herodotus appears to have

employed the word φθίσις also in this last named connection. We find Galen employing another word φθοή which is really the Attic equivalent of φθίσις , and in some of the works of Plato and also of Lucianus we find the word φθοή used with the same signification as φθίσις . Unfortunately the popular use of the word phthisis as equivalent to pulmonary tuberculosis has come to be recognised in some medical quarters, although most of those who adopt it are agreed that not all such cases are really tuberculous. It were well therefore if the word phthisis dropped out of use altogether as it tends to lead to endless confusion. It is most unfortunate that so great an authority as the late Sir Andrew Clark employed it, for it is greatly owing to his influence that the word phthisis has been retained in medical literature. Personally I prefer not to use the word at all, and when I do employ it I invariably understand it as signifying tuberculous disease of the lung, to be further defined as acute or chronic as the case may be. Had Clark confined his use of the phrase fibroid phthisis to tuberculous cases in which there was fibroid induration with contraction of the pulmonary tissue less ambiguity would have resulted, but unfortunately he included under it cases which would more correctly have been named

chronic pulmonary tuberculosis, for it is in this condition that we have fibrous tissue development taking place. Thus Fibroid Phthisis is nowadays used indiscriminately as applicable to pulmonary fibrosis which has had tuberculous changes grafted on to it and also to pulmonary tuberculosis in which fibroid changes have taken place. Thus it is apparent that the term Fibroid Phthisis is most ambiguous, possessing as it does a double meaning. There is no doubt whatever, as I have already pointed out, that many of the cases described by Clark and others under the term fibroid phthisis were in reality cases of chronic pulmonary tuberculosis, that is to say tuberculous cases in which a fibroid change had taken place subsequently thus leading to chronicity of the disease.

As regards the use of other names given to this disease we shall first consider that of Cirrhosis of the Lung. Doubtless this is a good enough nomenclature so far, but as it indicates that the disease has a similar pathogenesis to hepatic cirrhosis it is apt to prove misleading. Bastian uses it following the example of Corrigan and others, but all the same the adoption of this term is not to be commended. Again the use of the name chronic pneumonia, as adopted by Charcot, Andral, Wilson Fox and others, is most objectionable

as is also that of Interstitial Pneumonia, applied to this condition by Rotitansky, Juergensen and their followers. There is in pulmonary fibrosis no exudation such as occurs in a pneumonia, and in the majority of instances the disease arises quite apart from any pneumonic process. Juergensen, in fact, defines interstitial pneumonia as "the results of those inflammatory processes which take place in the connective tissue framework of the lung," (see Ziemssen's Cyclopaedia Vol. IX. p. 839), and thereby implies that the word pneumonia may be used indiscriminately, in other words that it may be applied to a process of connective tissue hyperplasia going on in the lung, as well as to inflammation of the lung substance. For similar reasons the long name chronic interstitial pneumonia should never be used, nor should that of fibroid pneumonia, more recently employed by Auld (vide Op. Cit. London 1892).

Handfield-Jones, believing the process to be really a degenerative one, gave it the unfortunate name of fibroid degeneration of the lung, regarding the change as dependent on a peculiar constitutional tendency of the tissue to degeneration of a fibroid nature. He looked upon fibroid degeneration of the lung as only one phase in a general degenerative process going on throughout

the body, and intimately related to hepatic and renal cirrhosis, cardiac sclerosis and degenerative changes in the nervous system. The other terms used at different times in connection with this disease need not further be referred to as they are now quite obsolete and are of purely historic interest.

Comparatively little literature has of late years appeared on the subject of pulmonary fibrosis, and as I have been fortunate in having to deal with several cases of this disease I am able to bring forward many facts of considerable clinical and therapeutic interest. I have already defined what is meant by the term pulmonary fibrosis, but I may pause here for a moment to emphasise more fully my interpretation of this nomenclature. Pulmonary Fibrosis is in the strictest and best sense of the term a primary condition, that is to say it has no relation whatever to tuberculosis of the lungs, although tuberculous changes may take place secondarily, and very commonly do, in a lung which is the seat of fibrous tissue hyperplasia. I admit that the term pulmonary fibrosis may with some degree of justice be applied to a tuberculous lung in which the affected area has become surrounded by fibrous tissue, but I fear lest by stretching the term pulmonary fibrosis to include

such cases ambiguity may arise. Accordingly I prefer to use it in its more limited application, and shall so employ it throughout this monograph.

The Etiology of the Disease.

In this monograph, as already stated, I do not include those cases which are due to tuberculous infection. Accordingly the etiological factors which I am now about to consider at some length will entirely exclude Pulmonary Tuberculosis as a cause.

We sometimes meet with a form of broncho-pneumonia in young children, more especially perhaps in connection with pertussis and measles, in which the whole process is subacute or even chronic in its nature. The inflammatory process in such cases is less rapid in its onset, course, and termination than in the acute form of this disease. Almost invariably there is even a greater tendency to collapse of portions of the lung tissue in these cases than when the process is a typically acute one. It must be remembered that we do occasionally come across children who are suffering form a subacute form of broncho-pneumonia for which no specific cause can be assigned. In every case tuberculous invasion is exceedingly

liable to take place. Failing this we sometimes find pulmonary fibrosis taking place. Fibrous tissue is laid down throughout the lung with the result that we get bronchiectatic dilatations with concomitant shrinkage of the affected organ. As a rule this fibrous tissue is deposited first of all in the interalveolar septa. A case of this kind was under my care some time ago in which the patient, a child of five years, was found to be affected with pulmonary fibrosis. He had had an attack of measles six months before, and at that time seems to have developed a broncho-pneumonia. He never got quite well, but ever since had had a nasty cough, and was getting appreciably thinner. On examination of the chest it was noted that expansion of the right lung was distinctly impaired, and that the upper part of the right side of the chest was depressed. On percussion, a certain degree of dulness was made out which extended right round into the axilla and also posteriorly. The breath sounds were very feeble in places, while in others the breathing was distinctly bronchial. There were no accompaniments save an occasional rhonchus just below the clavicle. The cough was most distressing, but only came on in paroxysms, while he was often free from it for hours together, and at times the patient had

attacks of breathlessness which always seemed alarming. There was distinct clubbing of the fingers with slight cyanosis of the lips and cheeks. This patient improved considerably under treatment, but unfortunately developed an attack of diphtheria to which he succumbed.

It is quite evident to any one who has taken the trouble to go into the subject for himself that not every case of broncho-pneumonia develops pulmonary fibrosis. Generally speaking, however, it seems fairly well recognised that many cases of broncho-pneumonia complicating measles or pertussis tend to become affected by this change. I have gone into the matter carefully, and have examined the chests of many children, who have had an attack of broncho-pneumonia following on one or other of these infectious diseases, and the conclusion I have come to is that in the case of pertussis we have a special liability to this hyperplasia of fibrous tissue, far more so than in that of measles. Why this should be so I do not quite clearly see at present, unless the mechanical irritation of the lung tissue set up by coughing plays a part in its production.

In this connection it is interesting to note how time and again we find that patients suffering from pulmonary fibrosis give a history of having

had "some lung trouble" in connection with an attack of measles or of pertussis in their early childhood.

Lobar pneumonia as a cause of pulmonary fibrosis is not in my experience very common. Still cases are occasionally met with in which the patient gives a distinct history of a previous pneumonic attack. Then again we have a separate disease in which a certain amount of fibrosis takes place, and which has been named very happily by Dr Percy Kidd in his valuable paper (vide Lancet, April 1890) Subacute Indurative Pneumonia. In this disease we find distinct post mortem evidence of fibrosis present, together with a marked tendency to cavity formation. One or more lobes may be affected by this fibrosis which specially involves the walls of the alveoli together with the peribronchial and perivascular connective tissue as well as the septa between the lobules. Cases of this kind usually succumb, but in some instances there can be no doubt that they become chronic and go to swell the list of patients suffering from pulmonary fibrosis.

Pleurisy as a cause of pulmonary fibrosis is even less potent than lobar pneumonia. Still cases are sometimes met with in the post mortem room in which we find enormous pleural thickening and adhesions, together with a marked extension of

fibrous thickening not only between the lobules but throughout the entire mass of the lung which may be very much contracted and compressed. Too much reliance, however, must not be placed on statements which we frequently find made, namely, that pleurisy per se is a common cause of pulmonary fibrosis. It is, in fact, almost always associated with more or less pleurisy; but this association does not necessarily imply that the latter disease is the real cause of the fibrosis, for as a matter of fact pleurisy itself rarely occurs apart from some affection of the lung tissue. Probably, therefore, in almost every case supposed to be due to pleurisy there has been present at some time or other disease of the lung substance itself. So too with pleurisy accompanied by effusion I am not aware that pulmonary fibrosis is often caused by this condition. True, in such cases a considerable amount of pleuritic thickening and adhesion undoubtedly remains, but I have never been able to satisfy myself that any of these cases ever developed a true pulmonary fibrosis. It is often stated, however, by writers on the subject that pleuritic effusions which have persisted for some time are apt to set up interstitial changes in the pulmonary tissue of the nature of a hyperplasia. I fancy, however, that in the few cases in which

this may have been found present after death the invasion of the lung itself by disease has been the real factor at work.

Empyema as a cause of pulmonary fibrosis is practically unknown. I have observed cases in which children suffering from empyema have shewn a considerable degree of contraction of the lung on the affected side after recovery had taken place, but such contraction must not be mistaken for pulmonary fibrosis, which is quite a distinct disease. Haemorrhagic effusions into the pleural cavity are rare, and probably never result in setting up fibrosis of the lung tissue. On the other hand malignant affections of the lung or pleura or of the mediastinal glands may, by the simple effect of pressure, bring about a considerable degree of pulmonary fibrosis. So too with thoracic aneurisms, which in some cases are responsible for its production, at least to a limited extent.

A more powerful factor in the production of pulmonary fibrosis than any already mentioned is chronic bronchitis. In such cases the chronicity of the disease is the essential cause. Patients who have had attacks of winter cough recurring for years and who are now perhaps beginning to feel that the cough seldom leaves them throughout the entire year, whose ankles are beginning to swell,

and the right side of whose heart is shewing signs of dilatation are those in whom we may expect to find evidences of a pulmonary fibrosis present. In one or two cases in which I have found this association of chronic bronchitis with pulmonary fibrosis there was a distinct alcoholic history, so that in all probability the origin of the fibrosis is not always to be put down merely to the bronchitis in these cases.

Round about a bronchiectatic dilatation we usually meet with a considerable amount of pulmonary fibrosis. This, in the majority of instances, is probably the cause and not the effect of the bronchiectasis. Still there are some competent authorities who think otherwise. This was the view adopted by the older clinicians who maintained that the interstitial tissue changes were quite unimportant. Corrigan was probably among the first to recognise the error of disregarding the pulmonary fibrosis which he endeavoured to prove was of first importance in leading to bronchiectatic dilatation. Still, in spite of this, we find more recent writers such as Hadley and Chaplin admitting that in a certain proportion of cases the fibrosis is undeniably secondary to bronchiectasis, although in such cases the hyperplasia is but slight in amount.

The irritation caused by the presence of a foreign body in the respiratory passages, if long continued, may readily enough set up interstitial changes in the lung together with a condition of bronchiectasis. Fracture of a rib, the broken end of which has penetrated the lung tissue, has in one or two well authenticated cases been responsible for the production of a localised pulmonary fibrosis. So too with other chest injuries. Such cases, however, are necessarily very uncommon, and I should not consider this a very important cause of the condition under consideration.

When we come to enquire as to the relationship which exists between syphilis and pulmonary fibrosis we are at once confronted with a mass of incontrovertible eveidence going to prove that syphilis is a potent cuase of this disease. As a rule, however, the fibrosis is localised, and is in such cases most marked in the neighbourhood of a gummatous infiltration of the pulmonary tissue. In hereditary syphilis the connective tissue of the lung is specially involved in these patients; and we find definite proliferation of the inter-lobular and inter-alveolar connective tissue taking place. It should be remembered, however, that many cases of so-called pulmonary syphilis occurring in children are really tuberculous in

origin, a fact which is extremely apt to be overlooked when we find evidences of fibrosis in a syphilitic child. In adults localised fibrosis around gummatous deposits is extremely common, but there is not wanting evidence to shew that in a few cases we find scattered all through the lung areas of fibrosis, while in one or two instances a more diffuse pulmonary fibrosis affecting the connective tissue throughout the entire organ has been met with. It must, accordingly, be granted that syphilis is not altogether unimportant as a causal factor in the production of pulmonary fibrosis.

Alcohol has been stated by one or two observers to be a cause of pulmonary fibrosis, probably on the analogy that it sets up cirrhosis of the hepatic tissue. I have gone very thoroughly into this question, and have not been able to bring forward any evidence to prove that alcohol, per se, leads to the production of pulmonary fibrosis. I shall presently consider alcohol in its relation to those pulmonic changes resulting from dust inhalation, and shall then express still more fully my views on this subject, but in the present place I may state that so far as I have been able to judge alcoholism, even of the most chronic type, is never a cause of pulmonary fibrosis.

I grant that many cases of this disease are met with in alcoholic subjects; but alcoholism has not, in my experience, been the only factor at work in these cases.. I well remember in this connection the case of a stone-hewer who had well-marked evidences of pulmonary fibrosis with thickened arteries and chronic kidney disease. For a long time I looked on him as a chronic alcoholic, in spite of his assurances to the contrary. At last I discovered quite accidentally from a near relative of his that I had entirely misjudged the patient who had unquestionably been an abstainer all his days. Since then I have always been on my guard against arriving at too hasty a conclusion in such cases.

I now pass on to consider the very vast and intensely interesting, not to say fascinating, question of the relation which dust inhalation bears to pulmonary fibrosis. My attention was early directed to this important subject by being brought into contact with a large body of stone-hewers amongst whom this disease was found to be rampant. Before speaking more particularly of stone dust as a cause of this disease I shall briefly refer to the potency of dusts of other kinds which lead to pulmonary fibrosis in our industrial classes throughout this and other countries.

The material inhaled may be either Inorganic or Organic in its nature, while the latter may be either of animal or of vegetable origin. It is the dusts of inorganic origin, which are generally speaking the most injurious. I shall consider each of these groups in turn, reserving my remarks on Stone dust till the end.

A. Inorganic Dusts. These are for the most part far more injurious in their nature than those of organic origin. They include stone dust, which is largely composed of silica and alumina and which produces the disease known as silicosis. They also comprise metal dust of various kinds such as are met with in a number of occupations, for example, steel grinding, diamond polishing, glass cutting, pottery making, millstone and crystal grinding, looking-glass polishing, and others. The disease produced by this last mentioned group of dusts is usually termed siderosis.

B. Organic Dusts. These may be either of vegetable or animal origin. I shall look briefly at each of these two kinds.

(1) Vegetable Dusts. These include flour and grain dusts of all kinds to which millers are exposed; a large group met with in factories of various kinds such as jute, cotton and tobacco; while they also comprise those dusts which are produced from wood and

by which carpenters and other workers of this description are liable to be affected. Lastly under this head must be considered those dusts which are due to carbon, the most important being coal dust which gives rise in coal miners to a specific form of pulmonary fibrosis termed anthracosis. This form of dust is also met with in other occupations such as in that of chimney-sweeps, locomotive engine cleaners and coalers. To a certain extent also carbon dust is inhaled by moulders, copper-smiths and others. I wish at this point to emphasise a fact which to my knowledge has never yet been placed on record, namely, that drapers who naturally inhale cotton dust suffer with considerable frequency from pulmonary fibrosis which is exceedingly liable to lead on to tuberculous changes in the lung. In support of this statement I know a family in which three of the members worked in a large drapery establishment, and all of whom developed pulmonary fibrosis which eventually led to invasion of the tubercle bacillus in every case. None of these patients inherited the tuberculous tendency, and all of them had previously to going into business led a healthy outdoor life on a farm in the south of Scotland.

(2) Animal Dusts. To this class belong those dusts which originate from the manipulation of wool, feathers, hair, and even of silk and leather; while horn and ivory dusts must be included under this group. Those who unload ships' cargoes are specially prone to suffer from the effects of wool and other similar dusts, and so too tanners and curriers as well as bedding manufacturers may become affected by the dusts peculiar to these occupations.

Such then are the varieties of dusts met with in the course of the various industrial pursuits, any one of which may produce pulmonary fibrosis. As already stated dusts of animal origin are far less harmful than those of vegetable origin; while it is the inorganic dusts which are most certain to give rise to pulmonary disease. We are now in a position to look for a little at the effect which the inhalation of coal dust has upon the individual. In coal mines a great quantity of this dust is thrown into the atmosphere and is inhaled by the miner who develops what is known as "the black spit". It has long been held that anthracosis, which is undoubtedly set up by this inhalation of coal dust, is a very common cause of pulmonary

tuberculosis. This view I cannot endorse, as personal observation and study of the subject have revealed to me the fact that coal-mining is at the present time one of the healthiest industrial occupations known, not only in this country but in all parts of the globe. It would also seem as if the inhalation of carbon was really protective and tended to lengthen the life of the worker. No doubt there is some truth in the fact that carbon is an antiseptic vegetable dust which really protects the worker who thereby tends to lengthen his life by his work. This view is corroborated by many medical men whose practice lies in coal mining districts, and who seldom if ever meet with tuberculosis amongst miners. This, to my mind, is a fact of much significance; and for my own part I regard the inhalation of coal dust as quite insignificant in the production of pulmonary fibrosis. When we come to examine a coal miner's lung we do not find much evidence of hyperplasia of the connective tissue, the only change of any importance being marked and almost universal pigmentation of the pulmonary susbtance.

Siderosis set up by steel dust inhalation is a very fatal disease, and carries off year by year large numbers of grinders in Sheffield and elsewhere. These men seldom reach the age of 40,

while not a few die at a much earlier period. In fact the knife grinder knows well that his occupation will eventually prove to be his death, but the fact that he has to live and the difficulty of finding other suitable occupation keep him at his trade until he eventually has to give in and sacrifice himself because of his work, which is at the best a bare struggle for existence.

The Pulmonary Fibrosis of Stone Masons More Especially Considered.

Thus far we have looked at the subject of pulmonary fibrosis from a general point of view, but I shall now consider it more particularly in relation to chalicosis or stone-masons' disease as it is sometimes termed. Throughout what follows I shall from time to time refer to pulmonary fibrosis in the more general acceptation of the term, but my remarks will have a more direct bearing on the disease as met with in stone-masons. As already pointed out I have been brought in contact with a very large number of these workers, and have consequently had exceptional opportunities afforded me of studying this disease as it affects this class of workmen. The results of my observations will, I trust, tend to throw some fresh light upon a disease which has many sad aspects, a disease

which in the main is amenable to treatment if the patient is seen early enough, and one which is daily cutting off many of those who have helped to raise the fabric in which some of us now live at ease, oblivious to the fate of the workers who made it possible for us to dwell in comfort and security.

The terms applied to this special form of pulmonary fibrosis are three in number, viz., silicosis, chalicosis, and pneumonokoniosis or pneumoconiosis of stone-masons. It is probably better to drop these names altogether, and employ the more general term of pulmonary fibrosis which indicates the essential lesion present in this particular disease.

The Etiology of Pulmonary Fibrosis Occurring in Stone-Masons.

Regarding the etiology of pulmonary fibrosis in stone-masons I have collected a considerable amount of information. The essential cause is undoubtedly dust inhalation. It is not builders who suffer from this disease, but stone-hewers. The term mason is applied to both, and builders always begin by stone-hewing, while during a bad season they very frequently take to hewing as a means of earning a livelihood. Consequently,

although builders as a class are seldom attacked by pulmonary fibrosis, it must be remembered that should they have to revert to stone-hewing they are quite as liable to contract this disease as those who have been hewers all their lives. Within recent years machinery has largely supplanted manual labour in the manipulation of stone work, but nevertheless there is a large number of men emgaged in stone-hewing throughout the country.

The character of the place in which the work of stone-hewing is carried on has a considerable influence in the production of the disease. Workers in covered sheds are more readily affected than those who hew the stone in the open air. In Edinburgh alone there are several of these covered-in sheds in existence which undoubtedly serve as death traps to those who unfortunately have to work in them. I have known more than one stone-hewer who has developed well-marked pulmonary fibrosis within six months while working in one of these sheds, although previously he was in perfectly good health. The dust rising in such sheds finds no outlet, and the worker is surrounded on all sides by the dust thrown off from the stone which he is hewing. When the work is done in the open air the dust is more readily disseminated, and is not inhaled to the same extent. In one of these sheds referred

to I have from time to time held a sheet of glass measuring 12 inches square, and on each occasion it has been covered by a deposit of dust to the depth of fully 1/16 of an inch within a period of something less than five minutes. This amount of dust, if accumulated for a like period all through the day of an avergare of eight working hours would mean a collection of .6 inches altogether; and as the workman probably runs the risk of inhaling a large quantity of this we can readily understand how his lungs become affected thereby. It is difficult to realise that the public health authorities should allow these sheds to exist; but unfortunately they still remain in our midst, though as they are generally placed in some secluded place they escape public observation altogether. I am becoming more and more convinced that if it were not for these covered sheds stone-hewers would be healthier, and would be much less liable to become affected by pulmonary fibrosis. I have certainly known stone-hewers who had previously worked in the open air without shewing signs of disease, and who owing to scarcity of work had perforce to labour in one of these sheds, within a short time begin to complain of one or other of the initial symptoms so characteristic of pulmonary fibrosis. Shed-workers, therefore, are in my opinion most liable

to contract this disease, and this must be borne in mind when considering how best to lessen the risks to which stone-hewers are exposed.

Another etiological factor of considerable importance is the character of the stone-worked. Building stones must be selected on account of their strength, durability and facility of working. Now the strength of a stone depends on its texture. Thus the crystalline elements composing it may be in close juxtaposition, or their arrangement may be looser. It also depends on its hardness and elasticity which vary greatly in different rocks. The structure of the stone will also influence its strength, according as it shews a confused crystalline arrangement as is well seen in granitic rocks, or a foliated, schistose arrangement exemplified in the mica schists of the North of Scotland. The durability of a building stone will be affected by its density or closeness of structure. A compact, fine-grained rock, such as sandstone, is not likely to be much influenced by attrition forces. The most durable sandstones are generally siliceous; and irregularly grained stones stand strain better then the more regularly grained ones. Calcareous rocks are not as a rule suited for building purposes. Building stones, moreover, will be more or less difficult to work according as they are hard or fracture easily.

The stone-hewer has accordingly, in different parts of the country, a variety of stones on which to work. Sandstones are usually composed of quartz or some highly siliceous mineral. There are quite a variety of these sandstones in the immediate neighbourhood of Edinburgh, and on these most of our stone-hewers are engaged. Then there is free-stone which cuts in any direction; there is also flag-stone which is easily split up into layers, and lastly there is micaceous sandstone which contains a large amount of mica. Another stone of sedimentary origin like sandstone is greywacké, more popularly known as whinstone. This is very abundant in the quarries around Peebles and Moffat, and in fact these two towns are built up of it. Limestones, also used for building purposes, are chiefly composed of carbonate of lime; and some of these are worked in Edinburgh also. Igneous rocks such as granite are composed of quartz, orthoclase or plagioclase felspar as the case may be, and mica. Granite is largely worked for building purposes, tramways, and other objects. It belongs to the acid group of igneous rocks and contains over sixty per cent of silica.

This slight reference to the nature and composition of building-stones is a very necessary digression, for unless we know accurately what is

the composition of the material worked how can we properly understand the effect produced on the pulmonary tissue by the products given off by these stones during their manipulation by the hewer? Generally speaking the finer the dust the more liable is it to be inhaled and to reach the lungs of the workman. Limestones and sandstones, therefore, are by far the most dangerous in this respect, because although they are less laborious to work than the harder granitic rocks they give off a finer dust, the particles of which are sharp and angular. The stone of which Craigleith Quarry is composed proved from its nature a most dangerous one to work, and most if not all of the workmen who hewed this stone, of which many of our Edinburgh houses are built, died at an early age, no doubt from pulmonary tuberculosis secondary to fibrosis of the lung tissue. No one can study this subject without being struck by the fact that Edinburgh hewers are practically all attacked sooner or later by pulmonary disease, and that at a comparatively early age. This is not the case with workers in other parts of the country. Accordingly the only inference we can legitimately draw is that the stones worked here give rise to a dust which is much more deadly in its effect than that given off by stones got in the neighbourhood of other large cities.

There are other influences, however, at work in the production of pulmonary fibrosis in stone-hewers. Thus their home surroundings are not always satisfactory. Living for the most part in badly ventilated houses, surrounded by a large family, and often having long distances to travel in all kinds of weather to and from their work, it is little wonder that these workmen are so readily attacked by disease. It must not be forgotten, however, that in some instances the home surroundings are perfectly good and comfortable. In fact I know of at least half-a-dozen stone-hewers who are affected with pulmonary fibrosis whose homes are in every respect clean and healthy. In the majority of instances, however, I think it may be granted that the home surroundings of this class of workmen as a whole are anything but desirable.

Then as to diet. These men have practically only one meal at home during the day. Many of them leave home without food at an early hour. Breakfast consists of a tin of tea heated (one should rather say boiled) over a fire, and a thick cut sandwich of bread with fried eggs. The midday meal is often but a repetition of this. At night they partake of a dish of sausages or bacon and potatoes followed by tea, all of which are usually served up in anything but an appetising form.

This menu is practically unvaried throughout the year, and I can say without fear of contradiction that the most elementary ideas of culinary hygiene are sadly lacking in most cases.

I have already referred incidentally to alcohol as a cause of pulmonary fibrosis; and though I do not for one moment regard it as a cause per se, still I think it must be admitted that the three factors which, linked together, play the most prominent part in the production of pulmonary fibrosis are dust, diet, and drink. Alcoholic indulgence by devitalising the bodily and mental powers of these men is bound to affect the lung tissue prejudicially. When we remember again that much of the alcohol taken is in the form of the worst possible grain whisky can we doubt but that this impure material will affect the consumer? I think, therefore, that alcoholic indulgence must not be altogether disregarded when considering the etiology of pulmonary fibrosis.

Regarding the previous health of the patient I am able to state from an analysis of fifty cases carefully investigated that only about ten per cent had any history of previous illness since childhood. Some of these men when they have been only a few years at their occupation are as perfect specimens of healthy subjects as could probably be got anywhere. The very nature of their occupation leads

to muscular development. I have obtained, however, a distinct history of chronic rheumatism in one or two cases, while in others a sharp attack of influenza seems to have immediately preceded the commencement of the pulmonary affection. Venereal disease of the nature of gonorrhoea or of syphilis could be traced in a large proportion of my cases, but I do not think that any one of them was in any way predisposed to pulmonary fibrosis on this account.

The family history is of great interest as shewing how far a tendency to pulmonary disease influences these cases. In no single case was I able to obtain a definite history of an hereditary nature. Many of my patients developed tuberculosis eventually, but none of them inherited it from their parents or relatives. The family history given in each of my fifty cases is not lacking in interest, at all events when these histories are collected together and summarised as in the following table:-

History.	Father Alone.	Mother Alone.	Father and Mother.
Rheumatism, or Rheumatism and Heart Disease.	5	3	3
Heart Disease with no Definite Rheumatic History.	2		
Chronic Bronchitis	1	2	

History.	Father Alone.	Mother Alone.	Father and Mother.
Renal Disease (probably associated with Heart Affection). .	2		
Indefinite Gastric Disorders. . .	2		
History of "Fits" (alcoholic?) . .	1		
Paralysis Agitans . .	1		
Malignant Disease:			
Uterus . .		1	
Stomach . .	1		
Brain . . .	1		

This table shews that a family history of rheumatism was obtained in eleven cases, that is in 22 per cent. This is a somewhat curious fact, and one which may seem of but little importance when the history of only 50 cases is recorded; but others have emphasised the relationship existing between rheumatism and pulmonary fibrosis, while some authorities maintain that cardiac lesions are not uncommonly met with in such cases. I, therefore, think that this table possesses a certain value. A family history of respiratory disease was only obtainable in five of my cases, and in each of these I think we may fairly assume that chronic bronchitis was the affection referred to in every instance. Chronic renal disease was not common in the parents, nor was gout or apoplexy ever met with. Cancer, however, was present in

the parents of three of my patients. Taking these histories altogether I must confess that, apart from rheumatism, there is little evidence that hereditary tendencies have much to do with the pulmonic changes met with in stone-masons, although doubtless there may be a diathetic tendency in the case of other classes of persons affected by this disease.

The Age at which Pulmonary Fibrosis Attacks Stone-Hewers.

I am not aware that any definite statement has ever been published regarding this important question. The practical bearing of this age-immunity limit, so to speak, is that could we definitely predict at what period of life the workman is likely to become affected by pulmonary fibrosis it might in some cases be possible to protect him by advising him to seek some other occupation before his pulmonary organs have become affected and his chances of securing other employment thereby materially lessened. The ages of my fifty cases at the onset of the disease, as far as could be ascertained, were as follows:-

Age.	No. of Cases.
28 . . .	1
29 . . .	0
30 . . .	0

Age.	No. of Cases.
31 . . .	1
32 . . .	0
33 . . .	1
34 . . .	0
35 . . .	2
36 . . .	3
37 . . .	5
38 . . .	11
39 . . .	13
40 . . .	6
41 . . .	3
42 . . .	2
43 . . .	1
44 . . .	0
45 . . .	0
46 . . .	0
47	1
Total	50

This table gives the average age of incidence of this disease as 38.34 years. The table further shews that by far the larger number of actual cases commenced at the ages of 38 and 39 years respectively with a distinct falling off after the fortieth year is reached. It must not be forgotten, moreover, that the casual cases met with

after this period probably would have shewn evidences of the disease had they come under observation earlier, but so far as I have been able to discover the first symptoms made their appearance at the ages stated in the table.

My observations go to prove that no stone-hewer may safely continue at his work after 35 years of age. To do so, even if he is in apparently perfect health, is to court disaster and to ultimately shorten his life very materially. I place what I choose to call the "critical epoch" of the stone-hewer's life between the ages of 35 and 40. Practically all who follow this occupation become affected at some time within this "epoch". Here, therefore, we find another practical hint as to prophylaxis, which if it could be readily acted on in all cases would do much to limit the occurrence of pulmonary fibrosis and its secondary results. This point will be again referred to when I come to speak of the prophylactic treatment of this condition.

Pathology and Morbid Anatomy.

Before discussing the symptomatology of pulmonary fibrosis I shall very briefly refer to its pathology and morbid anatomy, as the symptoms can only be appreciated when we have studied carefully the changes which this condition effects upon the

lungs and respiratory passages generally. The active agent in the production of the pulmonary fibrosis of stone-hewers is the dust which is inhaled during their occupation. This dust acts as an irritant. The particles inhaled are too numerous and varied for the ciliated epithelium, the phagocytes and mucous corpuscles of the respiratory passages to cope with, and the result is that these particles to a large extent find a lodgement in the pulmonary tissues, especially in the alveolar cells, and accumulate there. From the interalveolar spaces these dust particles pass by way of the lymphatics, and in this manner become disseminated. The effect of these particles is always to set up a great amount of bronchial and peribronchial inflammation. Together with this we find a certain degree of characteristic pigmentation. These dust particles stimulate the growth and proliferation of connective tissue throughout the lung. The alveolar walls grow, and as they grow they tend to obliterate the alveolar cells. This process extends gradually from one part of the lung to another until a whole area is thrown out of respiratory capacity. Not only is the connective tissue of the lung itself affected, but the pleura also becomes more or less thickened secondarily.

The general condition in which we find the lung is that of contraction which may be more or

less marked according to the extent of the fibrosis. As a rule the greater the amount of fibrosis, the greater will be the extent of the pulmonic contraction. This condition of contraction may affect the entire lung or only some portion or portions of it, the remainder in the latter case being increased in bulk by the presence of a compensative emphysematous condition of the lung tissue. When cut into the lung substance has a peculiarly tough and gritty feel, while its specific gravity is greatly increased. In most cases of pulmonary fibrosis occurring in stone-masons we find that although there may be a distinct predominance of fibrous tissue there is still left a certain amount of lung substance of considerable respiratory capacity. This fibrosis is usually best marked around the bronchi and in the interlobular spaces where it sppears as more or less dense greyish-white bands. This hyperplasia is also well marked between the lobules, and is frequently distinctly noticed spreading outwards towards the root of the lung, while the bronchial glands may even be encircled by an extension of this fibrous element.

There are practically always extensive pleural adhesions present, but in the majority of cases I do not think the thickening of the pleura which accompanies these is more than an accidental complication, as I have seen one or two cases in which

the pleural membrane was not increased in thickness to any great extent. Certainly it must not be taken for granted that when the pleural thickening is well defined the process of fibrosis has begun there. In pulmonary fibrosis arising from other causes than the inhalation of dust it is no uncommon occurrence to find the pleural surfaces closely bound together by fibrous adhesions. In the particular variety under present consideration we rarely find this adhesion so complete, and in the majority of cases of pulmonary fibrosis met with in stone-hewers there always exists more or less of a potential pleural cavity.

Scattered throughout the lung will be found nodules of a greyish-white colour, varying greatly in size, but generally small. These nodules are sometimes specially marked around the bronchi and in the perivascular spaces. These nodules are excessively hard, and if on the surface the pleura over them is found to be more or less thickened.

When we examine the lung tissue microscopically we find that there is abundant connective tissue proliferation and infiltration of leucocytes. The vessels are found to be more or less obliterated. This connective tissue growth is at first most marked along the lines of the interlobular septa, and is undoubtedly due to the absorption of dust particles from the alveoli. Wherever the irritation

set up by these particles is most intense there will be found the more marked fibrosis. At a later stage the bronchial walls tend to become surrounded by a mass of connective tissue. So too with the blood vessels which may thereby become obliterated in places. The intima and adventitia of the latter are apt to be thickened as well.

It very often happens that other conditions such as bronchiectasis are present in addition, but I need not enter into a discussion of their morbid anatomy as these are complications simply and not a true part of the original disease.

The Symptomatology of Pulmonary Fibrosis.

Here again I shall confine my attention entirely to the symptoms presented in that form of the disease as it occurs in stone-hewers. I have analysed my series of fifty cases with the view of discovering if possible the initial symptom or symptoms usually complained of by the patient.

Initial Symptom Complained of by Patient.	No. of Cases in which each Symptom was observed
Dyspeptic Symptoms, (mostly loaded sensation after food and sickness, with constipation) .	11
Weakness and Inability for Exertion	9
Dyspnoea	5
Vertigo	4

Initial Symptom Complained of by Patient.	No. of Cases in which each Symptom was observed.
Cough	4
Oppression, or Tightness in Chest	4
Vague Pains in Chest	3
Losing Weight, or Getting Thinner	3
Faintness, or Actual Syncope	3
Expectoration	1
Haemoptysis	1
Swelling of Ankles	1
Insomnia	1
Total	50

A brief survey of these initial symptoms may not be unprofitable. In my own series of cases the symptoms which were found to be most commonly present at the start were those relating to gastric disturbance of the nature of a chronic catarrh, and not far behind these came a feeling of weakness or of inability for any prolonged exertion. I am much impressed by this, as on reading text-books we find that the symptoms are usually stated to be these relating to the respiratory system as one would naturally expect. I am convinced, however, that when we find a stone-mason complaining of gastric trouble or of a feeling of bodily weakness we should at once ask ourselves the question, Is this patient's

lung not shewing early evidence of pulmonary fibrosis? It may not as yet be extensive enough to produce physical signs, but the symptoms mentioned should put us on our guard. Why, it may be asked, do we find gastric symptoms and weakness present at all in such cases? Is it not because the fibrosis which has already commenced is interfering materially with the proper action of the lungs and preventing imperfect aeration of the blood? Hence the stomach and tissues of the body generally are supplied with an impure blood, and in consequence they fail to perform their functions as before. Next to these symptoms come dyspnoea, vertigo, cough and a sensation of oppression or tightness in the chest. The two former I consider of much greater significance than the latter two in view of the fact that neither dyspnoea nor vertigo suggest to the physician's mind the possibility of pulmonary fibrosis. Dyspnoea when it occurs in stone-masons is stated by most writers to result from bronchitis, emphysema or a dilated right ventricle; but as, generally speaking, fibrosis is responsible to a large extent for the occurrence more especially of emphysema and of cardiac dilatation I think this symptom may fairly be taken as an early indication of the onset of this condition. Vague pains in the chest were complained of in three of my cases at the outset. In one instance the patient came

to me complaining of a catching pain over the left lower ribs. There was no friction present on auscultation, and little or no pain on pressure. He said the pain seemed to be deep seated, and as some months later he shewed marked evidence of fibrosis in the left lower lobe I am convinced that this pain may have been due to dragging on the lung tissue by the fibrous bands. Another patient said he felt pain over the left apex which darted through to the back and up to the shoulder of the same side. In this case also physical examination failed to reveal the presence of rheumatism or of respiratory disease. The third case in which chest pain was experienced, mentioned a feeling of intense soreness which was localised in the lower part of the right side of the chest, and had been treated by a medical practitioner in the country as "a liver pain". This patient also developed unmistakeable evidence of pulmonary fibrosis two years after I first saw him.

Patients, in my experience, rarely come because of their losing weight or getting thinner. Every one of the three cases included in the table given above had this symptom pointed out to me, in two instances by their wives and in the third by a friend. Naturally the patient himself is usually the last person to realise that he is losing flesh, and even when he does appear to notice this he will

not readily admit the fact. As a rule, however, this loss of weight is by no means a common early symptom of pulmonary fibrosis; rather is it one of the later manifestations of this disease. A feeling of faintness was experienced as the first symptom in two cases, while one patient actually gave the history of fainting fits on two occasions within a single week. Only one of my cases came at first complaining of troublesome expectoration; and even in his case there was little cough, while what there was occurred chiefly in the early morning. Haemoptysis brought one patient to consult me for the first time. Even from this patient by strict questioning I failed to obtain any history of the occurrence of the other symptoms already referred to. Oedema of the ankles, mentioned by one patient as being the first indication afforded him that anything was wrong, is not an early symptom; and as a matter of fact I cannot believe that this particular patient had not previously experienced other symptoms, though he constantly affirmed that his statement was absolutely correct. Insomnia, curiously enough, was the first symptom complained of by a patient who died within eighteen months after I first saw him. In his case the disease progressed very rapidly, a tuberculous invasion of both lungs as well as of the intestinal tract and other organs having taken place.

The Three Stages of the Disease.

Such then are the early symptoms of pulmonary fibrosis, a disease which must be regarded as most insidious in its onset, but progressive and unremitting in its extension and development. Looking at the condition in its wider aspects I think it may conveniently be divided into three fairly distinct stages from the clinical point of view.

First of all we have the Stage of Onset. I have already mentioned the initial symptoms, and need not further refer to them except to emphasise the fact that respiratory phenomena such as cough, dyspnoea and expectoration are by no means always the first evidences of this disease displayed by the patient. In this stage the patient is able to continue at his work with comparative ease. It is rare that we find the disease starting before the thirty-fifth year of life, and this stage may last for a very variable period, although I think three or at most four years is probably the average limit. I have, however, known stone-hewers who betrayed undoubted evidences of pulmonary fibrosis to remain in this first stage for a somewhat longer period, but in such cases the conditions under which they worked were exceptionally good, while enforced idleness during the slack seasons doubtless proved beneficial in arresting the progress of the pulmonary affection.

Sooner or later, however, the patient enters upon the second stage which I have called the Stage of Progress. If advice has not been sought before this stage is reached the patient is sure to seek it now. He continues at his work it is true, but he does so under difficulties. His chief complaints now are cough and dyspnoea, the latter being especially felt on exertion, while the former is most troublesome in the morning on waking. The presence of bronchitis will always aggravate these two symptoms, but as the fibrosis extends so as to involve more and more of the lung tissue the dyspnoea will tend to increase. Emphysema and dilatation of the right side of the heart will also intensify the patient's shortness of breath. It is the pulmonary fibrosis which is the origin of these secondary changes, and consequently it must be credited as being the fundamental cause of the dyspnoea. There is usually considerable gastric disturbance throughout this stage; and the patient tends to become pale, this pallor being most marked on the mucous membranes of the gums and palate. The face, owing to the outdoor nature of the patient's work, is usually red and weathered so that we must not judge therefrom as to the presence or absence of anaemia. As time goes on the patient has to rest from his work for varying periods. At these times

his complaint may be simply a condition of bodily weakness usually associated with dyspnoea, or he may have an attack of actual bronchitis. At other times we may find him suffering from an attack which may at first suggest influenza, and in which the patient feels distinctly ill, with a temperature of 99°. or 99.5°. Fahr., and in which his leading symptoms are weakness, distaste for food, and vague pains in the chest and back, associated with more or less severe headache. In such cases I think we have a slight inflammatory attack, the inflammation being probably situated around the fibrosed areas in the lungs. At all events the patient's cough becomes aggravated during such attacks, and I think the condition tends to progress much more rapidly afterwards. I cannot find any reference in the literature to the occurrence of such mild febrile attacks during the course of the disease as I have just referred to, but I think there can be no doubt about their actual existence as I have observed them several times in different cases.

During this stage various complications usually set in, the commonest being emphysema. Bronchitis, of course, is always more or less in evidence, while bronchiectasis may be found present towards the end of this stage of progress. Haemoptysis

may also be met with, although this, in my experience, is comparatively rare at this period. Should it occur the probability is that it is due to the presence of bronchiectasis, although it may very very rarely be due to tuberculous invasion. Dilatation of the right side of the heart is apt to become marked towards the end of this stage. After fighting in vain against the disease, working the while under difficulties chiefly in consequence of the dyspnoea which always becomes more marked as time goes on, the patient has at last to leave off work altogether. The duration of this second stage is rarely long. I think the outside limit might be placed at two or at most three years, and as a general rule it is considerably shorter. Much, of course, will depend on the conditions under which the patient lives and works.

As soon as the patient finally leaves off work he reaches the third stage, or what I term the Stage of Complete Breakdown. In this stage the patient is absolutely compelled to give in. Work is now quite out of the question. The picture presented by the patient who has arrived at this point in his journey is a very striking one. He is thin and haggard-looking, the once strong muscular arms and chest being now but faint shadows of their former selves, while the lower limbs and abdomen also display unmistakeable evidences of

wasting and loss of nutrition. The slightest effort, such as walking a short distance, gives rise to the most intense dyspnoea. The cough is always well marked, being distinctly worse in the early hours of the morning. There is also expectoration which, as time goes on, tends to become very abundant and may be streaked with blood especially if bronchiectatic dilatations are present. The emphysema is now usually very extensive, while the right side of the heart is found to be more or less dilated. Clubbing of the fingers and sometimes of the toes as well is nearly always present. The loss of weight is progressive, and in one or two cases we find hoarseness of voice, which may probably be due to extension of the fibrous change to the laryngeal tissue. This alteration in the voice was certainly not due to tuberculous invasion in the few cases in which I have observed it, and I cannot explain its occurrence otherwise. Cyanosis is often well-marked towards the end, and even apart from tuberculous infection we sometimes find diarrhoea setting in. In many cases, however, the lung becomes invaded by the tubercle bacillus which finds a most attractive resting-place in the enfeebled pulmonary tissue. It is usually during this third stage, and not before, that tuberculosis developes secondarily in these patients. I shall,

however, refer again to this matter at greater length.

This stage may last for a very variable period indeed. If the patient has given up work entirely before there is extensive involvement of the lungs so that he still possesses a considerable amount of active pulmonary tissue, then life may be prolonged almost indefinitely, especially if the patient is able to obtain abundance of fresh air and nourishing food. He may be thin and more or less dyspnoeic with chronic cough and expectoration, but unless some intercurrent disease sets in he may live many years a life of enforced idleness. On the other hand if the patient's lungs are seriously impaired, with probably the presence of bronchiectasis and serious cardiac dilatation, then life cannot be long extended; while should the tubercle bacillus gain an entrance the patient rarely lives beyond eighteen months or two years after he has given up his work.

The Condition of the Blood in pulmonary fibrosis has been carefully noted in nine of my cases from time to time. Generally speaking I found a more or less marked diminution in the haemoglobin and red blood corpuscles in these cases, with at times a tendency to slight decrease in the number of the leucocytes, especially of the polynuclear

neutrophiles. Poikilocytosis is frequently well marked, while in the very chronic cases this poikilocytosis may be extreme and associated with a striking reduction of the haemoglobin as well as of the leucocytes. The results of a few of my observations in the nine cases referred to are here tabulated.

TABLE/

Case.		Haemoglobin.	R. B. C.
A.	1st. Stage.	85%.	4,680,000.
	2nd. Stage.	65%.	4,100,000.
(T.B.)	3rd. Stage.	60%.	4,000,000.
B.	2nd. Stage.	62%.	4,200,000.
	3rd. Stage.	58%.	3,950,000.
C.	2nd. Stage.	50%.	4,000,000.
	3rd. Stage.	45%.	3,200,000.
D.	2nd. Stage.	80%.	4,300,000.
	3rd. Stage.	75%.	4,216,000.
	3rd. Stage.	72%.	4,000,000.
E.	2nd. Stage.	60%.	3,950,000.
	2nd. Stage.	58%.	3,680,000.
F.	1st. Stage.	73%.	4,050,000.
	2nd. Stage.	73%.	3,870,000.
G.	3rd. Stage.	55%.	3,800,000.
	3rd. Stage.	50%.	3,600,000.
(T.B.)	3rd. Stage.	50%.	3,650,000.
(T.B.)	3rd. Stage.	46%.	3,500,000.
H.	2nd. Stage.	75%.	4,500,000.
	3rd. Stage.	60%.	4,250,000.
(T.B.)	3rd. Stage.	49%.	3,960,000.
I.	3rd. Stage.	58%.	4,100,000.
(T.B.)	3rd. Stage.	53%.	4,020,000.

T.B. Signifies Secondary Tuberculous Infection.

Small Lymphocytes.	Polynuclear Neutrophiles.	Total Leucocytes.	Poikilocytosis.
26%.	65%.	6,500.	Not observed.
		5,300.	Slight.
		11,500.	Well marked.
		6,200.	Slight.
		6,250.	Fairly marked.
20%.	55%.	4,850.	Well marked.
		4,200.	Well marked.
			Absent.
			Slight.
		6,300.	Fairly marked.
			Fairly marked.
			Well marked.
		5,120.	Slight.
		3,980.	Slight.
		5,600.	Slight.
		5,600.	Well marked.
		8,400.	Well marked.
		13,200.	Well marked.
		5,950.	Fairly marked.
		4,800.	Well marked.
		13,500.	Well marked.
		5,600.	Slight.
		12,350.	Slight.

I am aware that objection may very reasonably be taken to any attempt made to draw conclusions from such a limited number of cases, but I think the following deductions may fairly be made:-

With regard to the haemoglobin there is no doubt that it tends to become more and more reduced as the patient passes from one stage of the disease to another. In the first stage it may range between 75 and 80 per cent, while in the second stage it may be as low as 50 per cent. Even in the third stage it rarely falls below 45 or 50 per cent. The number of the red blood corpuscles is often considerably reduced, and poikilocytosis is often very well marked more particularly in the third stage of the disease. It is from the number of the leucocytes that we obtain the most valuable information as to the progress of the disease. The number is often found to be considerably below the healthy standard especially during the first and second stages. During the third or terminal stage the number continues to be reduced so long as no secondary tuberculous infection takes place. When, however, the lung becomes invaded by the tubercle bacillus the blood examination reveals a very definite increase in the number of the leucocytes, more especially of the polynuclear neutrophiles. The largest number counted in any of my cases was 13,500; and

accompanying this leucocytosis was a considerable reduction in the haemoglobin percentage together with slight diminution of the erythrocytes which gave evidence of well marked poikilocytosis.

The fact of most practical value which I have gleaned from analysis of the blood in pulmonary fibrosis is that, even when repeated examinations of the sputum fail to reveal the presence of tubercle bacilli, their existence in the lung may be suspected should there be an increase in the leucocyte count; while, on the other hand, if the leucocytes are normal or still more if they are diminished in amount, we can be almost certain that no secondary invasion by the tubercle bacillus has as yet taken place. Accordingly my practice has recently been in every case presenting itself for treatment to examine, in addition to employing the ordinary physical methods, first the urine, and then the sputum, and if no tubercle bacilli are found in the latter to proceed to count the leucocytes, specially noting any marked increase in the polynuclear neutrophiles. In quite a number of cases I have found leucocytosis present when the patient was nearing the end of his journey, and although in many of these instances the sputum was apparently free from tubercle bacilli I concluded that pulmonary tuberculosis had probably set in.

At all events I now look upon marked leucocytosis as a grave sign in this disease, indicating, as it usually does in my experience, that the patient has not long to live.

The examination of the urine in these cases is usually limited to a determination of the presence or absence of albumen. In several instances, however, I have had the opportunity of studying the chemical composition of the urine more thoroughly, and on two of these I am able to report as follows:-

The output of phosphoric acid was diminished so that in one case it only reached 22.5 grains per day, while in another it was reduced to 20.75 grains. In neither of these cases was there any evidence of renal disease, nor was there ever albumen present in the urine. Both patients, moreover, were on ordinary diet which included a fair allowance of butchers' meat. The sulphates on the other hand were found to be considerably increased. The method adopted in estimating their amount was to compare the amount of precipitation obtained in a normal healthy urine with that got in the urine of the cases under observation. The test was carried out by first of all rendering the urine strongly acid by the addition of acetic acid in order to prevent the precipitation of

phosphates. Then a solution of barium chloride was dropped into a test tube containing exactly two inches of urine when a white precipitate indicated the presence of sulphates. The density of the precipitate so obtained as compared with that of the precipitate formed in the standard urine was taken as a measure of the increase or otherwise of the sulphates present. By this means I was able to recognise that there was always a slight increase in the amount of sulphates present.

It has been observed that increase of sulphates is sometimes found in the urine in leucocythaemia, but in neither of the patients referred to was there any marked alteration in the number or character of the leucocytes. As the sulphates of the urine are largely derived from the breaking down of tissue proteids I think this must have been the real cause at work here. Whether or not this increase of sulphates in the urine is to be met with in the majority of cases of pulmonary fibrosis I do not pretend to be able to state, but I think further investigations might be made with advantage in similar cases with a view to determining these points and also whether it is the ether-sulphuric or the free sulphuric acid which is increased.

Albuminuria was not met with in any of my cases examined during the first stage. Towards the the end of the second stage I obtained a trace (in three cases a good deal) in 15 per cent of the patients whose urine was examined. During the third stage a trace of albumen was rarely absent, although at times the urine was quite free from it. Towards the end examination of the urine almost always revealed its presence, while in a small percentage of cases I found a very large amount. In few instances, however, was there any marked alteration in the urea estimation. When secondary tuberculous involvement has taken place then I think we may almost expect to meet with albuminuria to a greater or less extent. In other cases when albumen is found in the urine its presence may be explained by the association of chronic renal disease with the pulmonary fibrosis. In the majority of cases, however, and especially when only a trace of albumen is found, it may fairly be assumed that it is simply due to renal engorgement and congestion resulting from impairment of the circulation. Albuminuria is not necessarily a grave sign, but its presence is always more or less apt to be associated with other conditions to which it tends to add its baneful influence. If present in large amount, and especially if associated

with oedema of the ankles and legs, there is little likelihood of the patient holding out for long.

Physical Signs.

The first question which presents itself for our consideration under this heading is which lung is attacked first; and this being determined what portion of that lung is singled out to bear the first evidences of the disease upon it. It is by no means easy to make dogmatic statements regarding either of these two points. In the first place the disease has often progressed considerably before the patient is first seen, so that we cannot readily tell where the fibrosis actually commenced. Again, this lesion may be associated with other changes such as emphysema, bronchiectasis and chronic pleural thickening, all of which make it extremely difficult for the physician to differentiate between the signs due to the fibrosis and those due to other concomitant lesions. All I have been able to make out is the portion or portions of lung involved when first I examined the patient. The results of these early physical examinations may be summarised as follows:-

Upper left lobe alone affected in .	4 cases.
Lower left lobe alone affected in .	14 "
Upper right lobe alone affected in .	5 "

Middle right lobe alone affected in	0	cases.
Lower right lobe alone affected in	10	"
Lower left and right lobes affected in	9	"
Upper and middle right lobes affected in.	1	"
Lower and middle right lobes affected in.	2	"
Greater part of both lungs affected in	2	"
Lower left, lower and upper right lobes affected in	2	"
Lower and upper left and middle right lobes affected in	1	"

From this table it is evident that in my series of fifty cases the part most frequently found involved was the lower left lobe (in28%). Next comes the lower right (20%) and close to it both lower lobes (in 18%). Regarding the upper lobes the right was alone involved in 10% of the cases, and the left alone in 8%. Less commonly we find other portions of the lungs affected at one and the same time. On the whole, therefore, I am inclined to think that pulmonary fibrosis most commonly begins in the lower lobes, the left one being most frequently attacked. Of course, another series of cases might afford evidence of quite a contradictory nature, but I think my series is a fairly typical one, and one from which fairly conclusive evidence may be drawn.

I shall now consider seriatim the evidences of pulmonary fibrosis as obtained from a physical

examination of the patient. It must be remembered that it is not always an easy matter in these cases to disassociate the signs due to fibrosis from those resulting from complications which are liable to occur in the course of this disease. I shall endeavour, however, as far as possible to state clearly the definite and unmistakeable signs due to the fibrosis alone.

Starting with inspection of the chest we find in the early stages of the disease a certain degree of limitation of movement. This is not always well marked, but as a rule close observation will detect it. In the later stages of course we not only find marked deficiency of expansion, but also retraction of the chest over a greater or less area. This retraction is due to actual shrinkage of the lung tissue itself. In the worst cases the affected part of the chest may remain almost, or even entirely, motionless during respiration. In advanced cases even the intercostal spaces may become deepened so that the ribs stand out boldly against the depressed spaces between them. A certain amount of lateral deflection of the spine is met with in some cases. Inspection, therefore, as a whole indicates that there is more or less diminution of the breathing capacity in the chest.

Palpation. As a rule I think palpation merely confirms what has been already found on inspection. In addition, however, by means of palpation we may be able to make out a distinct diminution in the vocal fremitus over the affected areas. In some few cases this is somewhat increased, but on the whole I think we usually find it diminished. In cases where the pleura is thickened we will, of course, have a still greater tendency for the vocal fremitus to be diminished. I have even found it absent altogether in patients who had as no great pleural thickening. It is generally stated that the vocal fremitus is increased in these cases, but this does not accord with my experience in the majority of cases at all events.

Percussion is often a difficult matter. We not uncommonly find a mixture of dulness, normal resonance and hyperresonance over different portions of one lobe. Then again the dull areas are not always accounted for by the presence of fibrosis, as a dull note may be due to other concomitant conditions such as inflammatory consolidation and much less commonly pleural thickening. There is, however, certain features which characterise the dulness due to pulmonary fibrosis. In the first place this dulness is often found to exist

more or less

in the form of more or less broad bands occurring in different parts of the lungs, but generally best marked toward the bases. Then again the note is more or less flat in character, and always accompanied by a distinct sense of resistance somewhat resembling that obtained over a pleural effusion. By this means we can distinguish an area of true pulmonary fibrosis from one of simple inflammatory consolidation.

If percussion is difficult, auscultation is still more so, as the almost inevitable presence of complications gives rise to adventitious sounds which tend to mask greatly the auscultatory signs due to the fibrosis alone. So much so is this the case that I think one must be a very expert auscultator before he can tell by this means alone that the area of lung tissue which he is examining is actually in a state of fibrosis. Under favourable circumstances and with a monaural stethoscope on listening over such an area we note that the breathing is of a feeble bronchial character or, if the fibrosis is extensive, absent altogether. There are no accompaniments beyond an occasional harsh creaking sound suggestive perhaps at first of pleuritic friction, but not altogether like it. The vocal resonance in uncomplicated cases is considerably diminished and at times absent. It may

even in some cases be increased, but when this is so we have probably more than a simple fibrosis present.

Such then are the physical signs of a pure case: but as I have already indicated it is seldom indeed that we do not find them considerably altered and masked by the presence of complications.

The Characters of the Sputum are of some interest. At first there is none, the cough being quite dry and harsh. Then the patient begins to expectorate a little greyish and very tenacious mucus which is always difficult to expel. The sputum is never purulent until we have bronchial catarrh superadded. As time goes on, however, the sputum becomes mucopurulent and eventually purulent. There is never any haemoptysis unless some complication occurs such as bronchiectasis or tuberculous infection. We may find particles of silica present in the expectoration, but elastic tissue and tubercle bacilli are never present in pure cases of pulmonary fibrosis. Even when tuberculous infection takes place the tubercle bacilli are rarely abundant. The expectoration is often most troublesome at night and in the early morning even when no bronchiectatic dilatation is present. Attacks of bronchitis occur from time to time, and and then the amount of expectoration is greatly

increased; while if bronchiectasis supervenes we get expectoration of a more or less typical appearance quite characteristic of the latter disease.

Pallor is often a sign of some importance in pulmonary fibrosis, but more noteworthy is the presence of cyanosis, the latter being a valuable indication as to the condition of the circulation. Clubbing of the fingers and toes is practically always got in this disease, and sometimes even the nose appears to be enlarged as well. The clubbing is always most marked in those cases associated with considerable bronchitis and emphysema or with bronchiectasis. It occurs, however, quite apart from these complications, and I have observed it in one case in the very first stage in which there could not possibly have been time for any emphysematous or bronchiectatic change to take place.

The State of the Heart is of very great importance in this disease, and indeed I lay considerable stress on the value of a careful cardiac examination when we are determining the prognosis of any particular case. I think it is always well to note carefully any enfeeblement of the heart sounds as well as any irregularity in the beats and strength of the pulse. There is no doubt that the dyspnoea in these cases is often rendered worse by the weakness of the heart, and consequently by examining the heart we may obtain a very valuable indication for treatment.

We often find a considerable amount of cardiac hypertrophy, more especially of the right ventricle. The right side of the heart, however, very generally shews unmistakeable evidence of dilatation; and towards the termination of the disease right-sided dilatation is almost invariably present. The heart can frequently be only imperfectly percussed out owing to the emphysematous condition of the overlying lung. Evidences of backward pressure are usually present towards the end, and oedema of the ankles and legs and even of the hands is commonly seen. Congestion of the viscera, especially of the kidneys and liver, the latter being frequently found to be enlarged during life, takes place as time goes on.

Displacement of organs is the rule. Thus the heart may be dragged over by the fibrosed lung to a varying extent, while the stomach, liver and spleen may all be dragged upwards with the diaphragm which follows the upward shrinking lung or lungs. It is often difficult to make out exactly how far this displacement extends owing to the presence of emphysema which renders differential percussion practically impossible.

The Complications Of Pulmonary Fibrosis.

As I have already indicated pulmonary fibrosis as met with in stone-masons rarely occurs alone.

Almost invariably it is complicated by one or more distinct pathological conditions. Of these by far the most frequently met with is Chronic Bronchial Catarrh. The presence of this is shewn by the patient's cough becoming increased in frequency and severity, while the expectoration becomes more abundant and from being mucoid simply it changes to muco-purulent. These attacks of bronchial catarrh become more and more frequent as the disease progresses, and have a very deleterious influence on the patients general condition. Every fresh cold means a fresh catarrhal attack and the risks of these to the patient are enormous. In the first place they facilitate not only the entrance of the tubercle bacillus but even its spread throughout the lungs. In the second place it must not be forgotten that an emphysematous condition is almost certain to result from prolonged bronchial catarrh, apart altogether from its being induced by the pulmonary fibrosis itself.

Emphysema is also present to a greater or less degree in almost every case. It may be brought about by the fibrosis itself, and is then of the nature of a compensative change. On the other hand it is not infrequently the result of chronic bronchial catarrh. In whatever way produced emphysema sadly hampers the patient's respiratory

powers. The lung tissue loses its elasticity, the capillaries tend to become obliterated and the right side of the heart dilates eventually, while backward pressure phenomena tend sooner or later to manifest themselves especially by swelling of the ankles and cyanosis of the face. In pulmonary fibrosis we usually meet with the emphysematous areas in the upper part of the chest, more especially perhaps on the left side over the praecordial region and towards the apex, though it may also be found quite as extensively on the right side of the chest. It is rarely met with over the lower lobes posteriorly. When emphysema occurs in the chest of a patient suffering from pulmonary fibrosis we rarely find the typical barrel-shaped condition so commonly associated with the emphysema due to chronic bronchitis and which is so well exemplified in labourers suffering from this affection. In pulmonary fibrosis accompanied by emphysema the chest is always more or less hollowed or retracted, the fibrosed tissue acting as a check to the emphysematous portions of the lung which would otherwise tend to bulge. I have never up to the present met with a stone-hewer who had a barrel-shaped chest, and accordingly I believe my statement on this point to be correct.

Another complication of very great importance

is Bronchiectasis. The origin of bronchiectatic dilatation in cases of pulmonary fibrosis is not quite obvious. It may be that the fibrosed tissue causes dilatation of the bronchi in its immediate neighbourhood by the simple strain which it produces. Another cause, suggested by Laennec, is that the retained secretion in the bronchi causes dilatation to take place; but we have little evidence to shew that the secretion is actually retained in sufficient amount to produce anything like a dilating force on the bronchial walls. At the same time we must not too readily admit that the fibrosis is the sole cause of the bronchiectatic dilatation in these cases. Probably in this disease the walls of the bronchi are weakened by repeated catarrhal attacks so that they yield more readily to the dilating strain exerted upon them by the fibrosed tissue around them. Bronchiectasis usually appears towards the end of the second stage of this disease, or if it does not come on then it is seldom long delayed when the patient enters upon the third stage. Breathing then becomes more embarrassed, and the cough is aggravated, while the expectoration becomes more abundant, a noteworthy feature being that the patient has paroxysms of coughing in the early morning during which he brings up a large quantity of

putrid, foul-smelling sputum frequently blood-tinged. These paroxysms generally moderate in severity as the day wears on. It is not always an easy matter to recognise bronchiectasis in these cases by physical signs alone, but generally speaking bronchiectatic dilatation should be looked for towards the bases of the lungs rather than in the neighbourhood of the apices.

In speaking of the complications of pulmonary fibrosis we must not omit to mention Pleurisy. It must not be regarded, however, as of very common occurrence. Its presence is often erroneously diagnosed because the practitioner hears a peculiar sound over a certain portion of the chest. This sound is characteristic of pulmonary fibrosis and is of a somewhat creaking character, but is really not the same as the friction heard when pleurisy is present. I am afraid it is difficult to describe this peculiar sound exactly, but when once heard it can scarcely be mistaken again. Pain too may be present, although pleurisy is not the cause. This pain in all probability is due to the contracting process taking place in some part of the fibrosed area of lung tissue. If pleurisy does occur a strong suspicion should be felt that secondary tuberculous infection of the lung has taken place.

Acute Lobar Pneumonia formed a complication in one of my cases. It came on in the second stage of the disease. The pneumonic process was found over the lower and middle lobes of the right lung. The temperature tended to be somewhat irregular, and the crisis did not occur until the tenth day. Six months later tubercle bacilli were found in the sputum, and the patient died soon after. Pneumonia, however, cannot be regarded as being a usual complication, or even one which may be expected to supervene. Its occurrence must inevitably still further weaken the pulmonary tissue and render the soil more suitable for the growth and development of the tubercle bacillus.

Collapse of Portions of the Affected Lung occasionally occurs; while Acute Pulmonary Oedema may take place with startling suddenness. I am inclined to think that the latter was the real cause of death in one of my cases, but as no post-mortem examination was made I am unable to state this with absolute certainty.

Haemoptysis may be met with in two forms, either as a mere streaking of the sputum, in which case a certain amount of bronchiectasis may be present, or in a more copious form and here secondary tuberculosis is more usually the cause. I remember one patient, however, who had a fairly

severe haemoptysis which lasted for three days. I could never detect tubercle bacilli in the sputum, nor could I find albumen in the urine, nor any leucocytosis, although I examined the blood on several occasions. The patient made an excellent recovery, and is still alive, although the attack referred to occurred fully four years ago. The presence of blood in the sputum, however, is not a good sign; although a mere streaking of the expectoration does not necessarily indicate any immediate danger.

I have never met with Gangrene of the Lung in patients suffering from pulmonary fibrosis, though it may occur as a complication of this disease. It is, I think, more likely to supervene in cases where there is great bronchiectatic dilatation and marked tuberculous invasion. This is of course a grave complication, and must certainly end in death soon after the diseased lung becomes gangrenous.

The occurrence of Waxy Changes, more especially in the liver, spleen, kidney and intestines may be associated with pulmonary fibrosis. Apart from this, however, we not infrequently find the liver enlarged from other causes such as congestion. It may also appear to be enlarged when in reality it is merely pushed downwards by an emphysematous lung. In rare cases it may be enlarged from

actual cirrhosis. Waxy disease of the intestines may give rise to troublesome diarrhoea, and I have found this to be a fairly common complication in the third stage of pulmonary fibrosis. So far as I can gather diarrhoea in these cases is generally looked upon as an indication of tuberculous invasion; but I am not prepared to admit that this is really the cause in most cases. It is generally due to waxy changes which are entirely dependent on the chronic fibrosis of the lung tissue. Diarrhoea is not a favorable complication, as if long continued it serves to still further weaken the patient. It is more apt to occur in those patients who are not over careful in their dietary, and also in those who have a tendency to constipation a condition which of itself produces irritation of the intestinal mucous membrane.

I have already mentioned Cardiac Dilatation as a complication of pulmonary fibrosis, and to this may be added hypertrophy which is usually present at some period of the disease. The right side of the heart is more affected as a rule than the left; and accompanying the dilatation may be considerable cyanosis, venous pulsation in the neck, and even oedema of the ankles, while the pulse may even be feeble and somewhat irregular. The dyspnoea is also apt to be increased; and

altogether cardiac dilatation greatly aggravates the patient's condition.

The Relation of Pulmonary Tuberculosis to Fibrosis.

The popular belief that every stone-hewer dies of pulmonary tuberculosis needs to be modified to some extent, for although many no doubt do become eventually infected by the tubercle bacillus, there are others who do not. The term "phthisis", as I pointed out at the very beginning of this monograph, is quite a misnomer when applied to these cases of pulmonary fibrosis. The name pulmonary fibro-tuberculosis may, perhaps, be legitimately given to those cases of fibrosis which become secondarily affected by tubercle, but we must be careful regarding our use even of this term which should be reserved solely for such cases as shew unmistakeable evidence of tuberculous invasion of the fibrosed lung tissue.

It was stated by Ludwig Hirt nearly a quarter of a century ago that something like over thirty-six per cent of stone-cutters, including masons, die of "consumption". It is exceedingly difficult to obtain reliable statistics on this question as many cases which are probably pure fibrosis are certified at death as "phthisis", "chronic bronchitis", "haemoptysis" or some other condition. I

am confident, however, that Hirts' figure is fairly accurate; although it is to be greatly regretted that more accuracy is not observed by the profession in signing the death certificates in these cases.

The invasion of the affected lung or lungs by tubercle bacilli usually occurs during the third stage of the disease, though in some instances it may occur much earlier. A good deal doubtless depends on the family history of the patient, and the occurrence of pulmonary tuberculosis in his parents or near relatives, although none of my cases afforded a history of this kind. It is sometimes thought that the mere fact of the patient inhaling dust and working under most unhygienic conditions is sufficient to account for this tuberculous invasion. I do not, however, think this is a sufficient cause for its occurrence. I am convinced that the real cause is that we have a lung weakened by fibrous change and associated in the majority of cases with a catarrhal condition of the bronchi. Under such circumstances the tubercle bacillus having once effected a ready entrance rapidly multiplies and disseminates itself throughout the diseased organ.

The character of the disease very often shews a marked change when secondary tuberculous invasion

has taken place. Frequently we find that one or both apices, which perhaps previously shewed merely the physical signs of emphysema, now give evidence of tuberculous consolidation by the presence of dulness associated with abundant moist sounds. In some instances we find these changes taking place at one or other base. Very often an attack of pleurisy gives us the first hint as to these fresh changes taking place. In any case the cough now becomes aggravated, while the expectoration will tend to become increased and to be more constant during the day than before. Haemoptysis will sooner or later set in, and tubercle bacilli will now for the first time appear in the sputum. In the most advanced cases as a rule these bacilli will be present and readily found on examination. In some instances, however, repeated staining of films fails to detect them. In such cases I rely for my diagnosis to a large extent on the examination of the blood and of the urine. The former nearly always shews a marked leucocytosis. It is the polynuclear neutrophiles which are specially increased in number under such conditions, while the haemoglobin will be diminished, and other less characteristic blood changes will be present. Thus in one of my cases I found that the haemoglobin had fallen to 49 per cent, and the red blood corpuscles, which shewed well-marked poikilocytosis, had been

reduced to 3,960,000, while the leucocytes numbered 13,500. In other cases the haemoglobin percentage varied between 46 and 60, while the red blood corpuscles numbered from 3½ millions up to a little over 4 millions. In most instances there was well marked poikilocytosis, although in one case it was only slight.

The urine in these cases is almost always albuminous, although before it had been found to be perfectly free from albumen. Of course the presence of albumen does not by any means always indicate tuberculous invasion of the lung, but it is suggestive of its occurrence in most cases. The amount of albumen may be small, but in one or two of my series of fifty cases it was pretty copious.

Once the lung becomes invaded by tubercle the process tends to spread more or less rapidly so that both lungs tend sooner or later to become riddled throughout. Sometimes we find both lungs affected almost simultaneously. The apices form the starting point as a rule, but not always as basal tuberculosis is by no means unknown to occur in the pulmonary fibrosis of stone-masons.

Pulmonary fibrosis is practically an afebrile disease, although as I have previously pointed out we meet with occasional slight febrile attacks in some cases. When tuberculosis ensues, however,

the temperature chart soon assumes its characteristic hectic appearance, and night-sweating becomes more or less marked. The emaciation now becomes extreme, and the patient, thin and wasted before, now appears as a mere skeleton. If confined to bed he usually developes bed-sores, while diarrhoea and extremely suffocative attacks of dyspnoea are apt to supervene. After tuberculous invasion has taken place life is seldom prolonged beyond a few months, although in a few exceptional cases the patient may, if placed under favorable surroundings, live on for a period of one or even two years.

In one or two instances I have found pneumothorax present a few days before death and in these cases the dyspnoea was very urgent, the patient being unable to lie down and calling out constantly for air. As a rule the pneumothorax is limited in extent when it does occur in pulmonary fibrosis associated with tuberculous lesions, but it is none the less liable to produce distressing symptoms and to cut off the patient within two or three days at most after its occurrence.

Pulmonary tuberculosis is, therefore, to be regarded as by far the most serious event that can take place in the history of a case of pulmonary

fibrosis. Its occurrence practically means that the patient is placed beyond all hope of prolonging his days, as in these cases no one can tell how soon he may be cut off, death sometimes occurring with startling suddenness while the patient is going about and his friends complimenting him on his apparent improvement.

Progress and Duration of the Disease.

Pulmonary fibrosis is not necessarily a disease which of itself tends to greatly shorten life. Were it not for the fact that complications are bound to occur sooner or later the stone-hewer would probably live to a much longer age than he does at present. The disease, starting as it does towards the end of the third decade of life, at first, as a rule, progresses somewhat slowly and the patient in many instances is able to remain constantly at work; while even in some cases he is not apparently conscious that anything is wrong. Dyspeptic symptoms or a feeling of bodily weakness may after a time display themselves, but on the whole the patient at the start is not to outward appearances an invalid. Slowly but surely, however, the disease progresses bringing in its train phenomena which are more definitely respiratory in origin, such as cough and dyspnoea. At times, it may be, the patient has to stay off work on account of

bronchitis or a feeling of more than ordinary weakness. When the second stage is reached slight febrile attacks are not at all uncommon, while during this stage all the symptoms tend to become more marked. Eventually, after having a series of enforced rests from work, the stone-hewer has to take a rest which proves to be the final one until death claims him as its victim. Very often when the patient gives up work for the last time, and enters upon the third stage of the disease, he imagines that as before he will resume his employment after he has had a few weeks' rest and treatment. In this hope he is destined to be ruthlessly deluded. He now goes rapidly downhill, and one or other of the complications already referred to is bound to set in. The latter not only add to his discomfort and distress, but they very materially influence the downward progress of the patient. This is more especially the case when tuberculosis supervenes, as then but a few months must elapse ere the patient's life is ended for ever.

The age at death varies considerably, but between the ages of 39 and 42 we probably find most deaths taking place from pulmonary fibrosis or one of its complications. Thus in twenty-seven deaths occurring within my own observation I find the ages represented as follows:-

Age at Death.	No. of Cases.
37 . . .	2
39 . . .	2
40 . . .	5
41 . . .	8
42 . . .	5
43 . . .	3
44 . . .	1
46 . . .	1
Total	27

Certainly I think few, if any, stone-hewers will be found alive after the age of fifty, which is in my experience too great a limit to place upon the life of this class of workmen. This is a matter of great practical importance from the point of view of life assurance. Those companies which do a large industrial class of business necessarily have many proposals from stone-hewers. The sums assured are never large, it is true. In fact they seldom range above £30, and in rare cases extend to £50; much more frequently the proposals on the penny a week system do not cover a greater risk than £10. Still even this very small risk should not be accepted by any company if the proponent is over twenty-five years of age. At twenty-five years no doubt the stone-hewer is a perfectly healthy man,

but by the time he reaches the age of thirty-five his chest may have become affected, while at forty he will more than likely have but a few months to live.

Another aspect of the question is brought into view when we come to consider the admission of stone-hewers into workmen's societies. These would soon become bankrupt if composed entirely of masons. I have been told by the secretary of one of the most popular of these societies, which is largely made up of the mason element that nearly all the deaths during the year occur in this class of workmen, while it is these men who are constantly draining the funds by habitually remaining on the sick list.

It is generally stated that pulmonary fibrosis is a slowly progressing disease. This is not the case so far as my experience goes when it is met with in stone-masons. Within the last four or five years I have had several patients under treatment who shewed but slight evidence of pulmonary disease at the start, and who have died within two years, often less, of their being first seen. One fact I can state very dogmatically and that is that, provided the workman can give up work entirely while the disease is still in the earlier part of the second stage, he may live on for quite a number of years. I have two or three such cases

at present coming under my occasional observation who were fortunately enabled to take my advice and permanently give up work so soon as a definite diagnosis had been made. Accordingly I am strongly of opinion that it is quite possible to arrest the progress of the disease under favorable circumstances, but the patient's strict abstinence from stone-hewing is a sine qua non. Even when the patient cannot entirely live a life of idleness he may, by obtaining some other suitable occupation, be enabled to prolong his life. Unfortunately stone-hewers are not on the whole an intelligent class, and it is not therefore by any means an easy matter for them to find other employment. Some of them find their way into rubber factories, but needless to say such a change of work is far from beneficial. Outdoor work of some kind is best, but this can seldom be had when wanted. If the patient must continue at work until he is forced to give it up altogether then his life will be indeed a short one, and his closing days will often be made intolerable by the presence of intense dyspnoea and a cough which seems to rack the whole body by its severity.

The Mode and Cause of Death.

In a large proportion of cases death occurs with comparative suddenness. In only two of my

potent in bringing about a fatal result has been credited as being the actual cause of death. Thus in nearly all the cases in which a tuberculous element was superadded symptoms of heart failure were present as well as those of bronchiectasis. It is, therefore, not always an easy matter to assign the cause of death to any one particular pathological condition. In signing the death certificate I have invariably stated pulmonary fibrosis as the actual cause of death, merely mentioning the presence of tuberculosis if such existed. I think this is much more accurate than to give pulmonary tuberculosis as the cause of death, when in reality this is merely a complication and not actually the primary disease.

In a number of cases I have found a few days before death an aggravation of the dyspnoea, slight oedema of the ankles, and a well marked malar flushing associated with, it may be, slight cyanosis of the lips. The patient under such circumstances will frequently be going about, and even feeling better, perhaps even not coughing so much as hitherto. His friends will often tell you he is taking his food better, and is hoping that it will not be long now before he is back to work again. I never feel very certain of the patient when these signs are present, and usually warn the friends that the

end may be approaching more rapidly than they think. Death, I have said, usually takes place with startling suddenness in these cases. One of my patients shewed some of the evidences of approaching death I have just referred to, but neither he nor his wife could be got to believe that he was in danger. I saw him at 4 p.m. one afternoon. He had taken a good tea and said he felt much better than he had done for months. At night his wife went to bed, lying at the foot. She woke up about 3 a.m., and felt something cold touch her cheek. It was her husband's feet. He must then have been dead some time as the body was quite cold. This case illustrates well how unexpectedly death may come to these patients. This man had the peculiar bright red cheeks and the slight cyanosis of the lips which I never like to see, and which always serve to indicate to me approaching death or at all events that it is not very far off.

Death, however, may be preceded by agonising dyspnoea. Probably some of these cases have developed a pneumothorax, but only in two of my series was I able to definitely make out its occurrence. It is no uncommon sight to see these patients a few days before death sitting up in bed, supported by pillows, and gasping for breath as if each respiratory act was destined to be their

last, the cold perspiration standing constantly upon the forehead of the sufferer. So intense does the dyspnoea become in these cases that it is often found quite impossible for the patient to take any food. Thirst is frequently a troublesome feature, and attempts at swallowing water or other liquid may cause a fit of choking and coughing.

There are thus, broadly speaking, two modes of death in pulmonary fibrosis. There is the form of death which takes place quite suddenly, often without any warning whatever of its approach, and there is the somewhat lingering form in which the patient is more or less completely bedridden for some days, or it may be weeks, before the end. In the majority of my cases death has come on in a more or less sudden fashion, and I have often examined a patient one day who was going about, with difficulty it is true but still able to take his daily walk, who was dead the next.

Prognosis.

After what I have already said there is little left to state under the heading of prognosis. Cure is absolutely impossible; but the disease may be arrested provided the patient can be got to leave off stone-hewing early enough. The prognosis, therefore, will be greatly influenced according as the patient gives up his occupation or continues at

it. Pulmonary fibrosis, pure and simple, is quite compatible with prolonged life, but unfortunately when it attacks stone-masons it brings various complications in its train, each and all of which materially affect the ultimate prognosis of the disease. Pulmonary tuberculosis, when it occurs secondarily, makes the prognosis very grave indeed. Any particular case in which we have to consider the prognosis at the time must be regarded mainly, therefore, from the following aspects:-

(1) The extent of the fibrosis itself.

(2) The nature and severity of the complications.

(3) The intensity of the dyspnoea.

(4) The surroundings of the patient, and whether or not he has given up his work entirely.

The habits of the patient must also be taken into account, as an intemperate man has little chance of holding out long when affected by this disease. If the fibrosis is slight in extent the dyspnoea not well marked, and the patient able to give up work entirely then the prognosis is not at all bad. Such a patient may live for a fairly long term of years. The gravest complications are undoubtedly emphysema, bronchiectasis, cardiac dilatation and pulmonary tuberculosis. The onset

of diarrhoea and of waxy disease renders the prognosis less hopeful. The examination of the urine and of the blood too must not be left out of account in estimating the prognosis. Marked diminution in the various elements is not incompatible with life, but a leucocytosis is not a hopeful sign. So too constant and especially increasing albuminuria must be looked upon as indicating death at an early period.

Haemoptysis may be specially referred to in connection with the prognosis of this disease. It is usually regarded as a grave sign. No doubt the physician does not look upon it favorably; but at the same time too much stress must not be laid upon its occurrence. Streaking of the sputum may be present for a considerable period without apparently affecting the prognosis in any way; and in one of my cases a copious haemoptysis occurred some years ago, but the patient is still alive. In some cases, indeed, I regard a slight haemoptysis as beneficial in relieving the backward pressure. Epistaxis sometimes occurs in this disease, and is helpful in like manner.

It is not easy to foretell how long the patient will live in any particular case; but extreme dyspnoea, marked albuminuria, feeble and rapid pulse, and swelling of the ankles must be

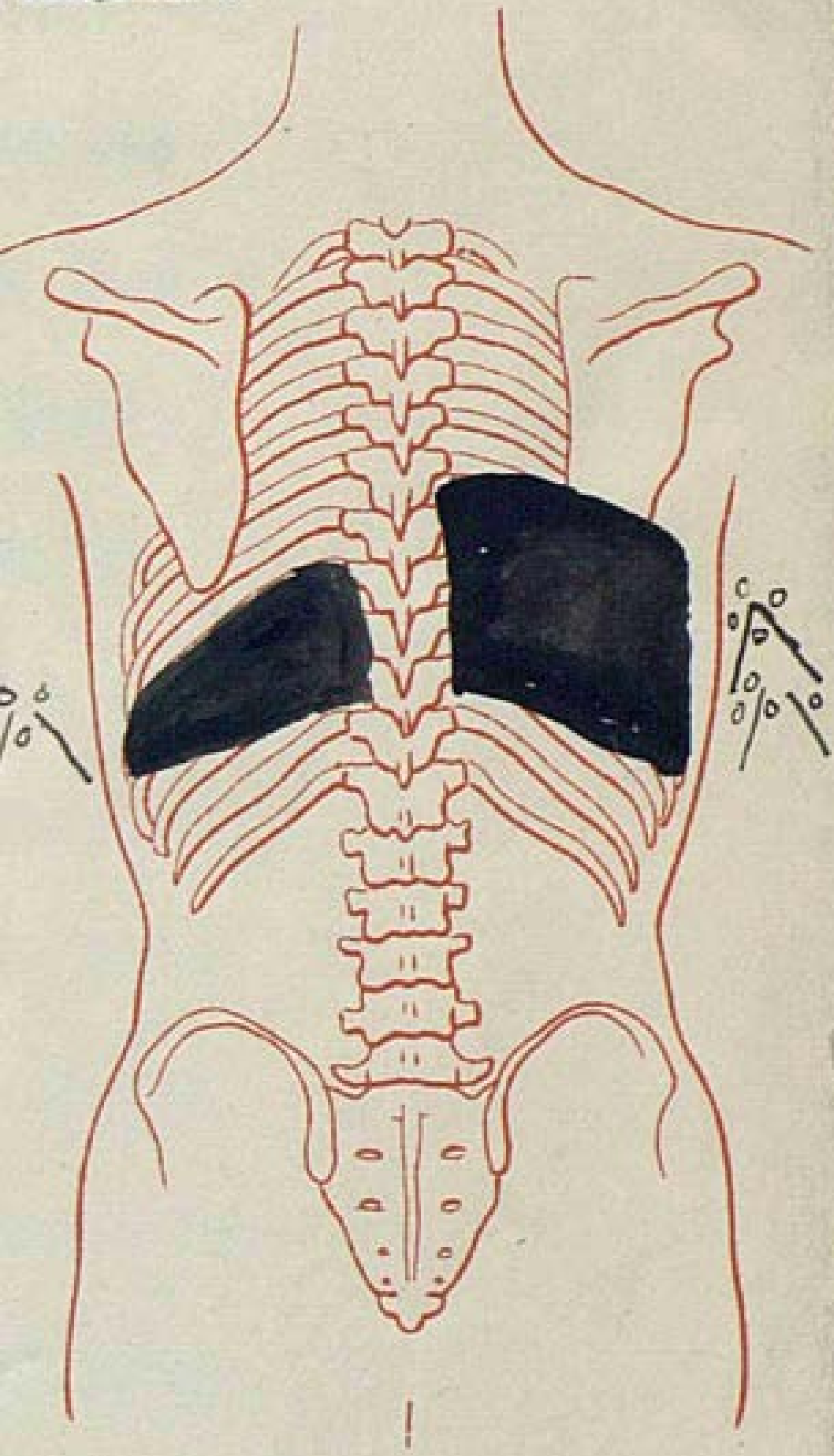
considered as indications that the end is fast approaching, even though the cough lessens and the patient is thought by his friends to be improving.

Brief Histories of a few Actual Cases.

I have thought it advisable to illustrate what has gone before by giving a few very limited outlines of some of my own cases. It is not possible to give them all, nor yet to go into any of the cases very fully; but I shall indicate the salient clinical points in each. In connection with most of those described I have given a self-explanatory diagram on which is mapped out the areas of lung tissue involved together with figures shewing the various auscultatory signs obtained over the affected areas. The majority of these diagrams were filled in on the first occasion of the patient's seeking advice. They are as nearly accurate as it was possible to make them, for it must be remembered that physical examination is by no means an easy matter in many of these cases.

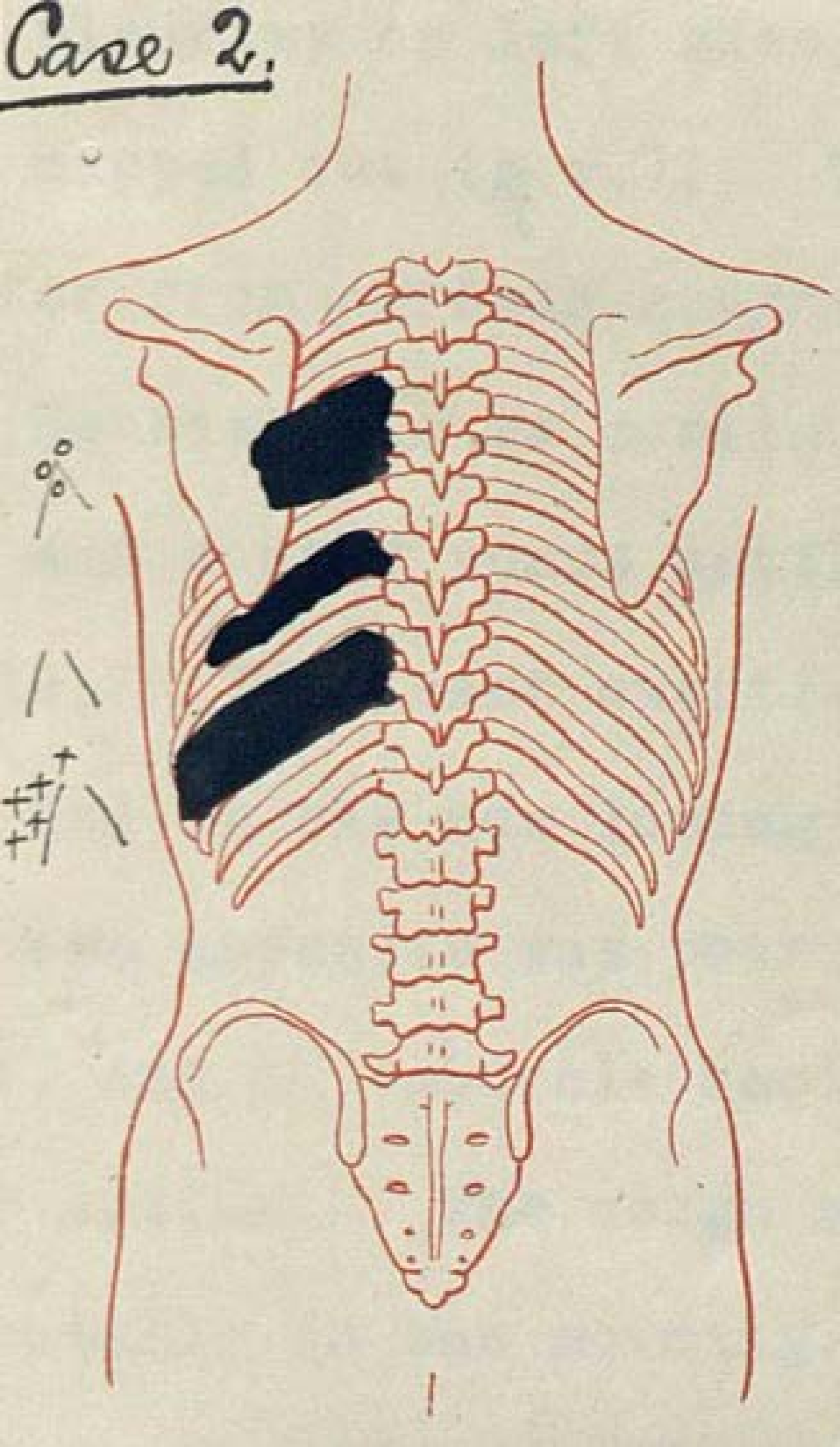
Case I. J.S., aet. 39, married. Had been a stone-hewer all his life. Very alcoholic until recently. Home surroundings fair. Father died of heart disease, Mother of uterine cancer. Complaint cough and shortness of breath. Duration "since an attack of influenza five months before". Both bases affected. Considerable emphysema.

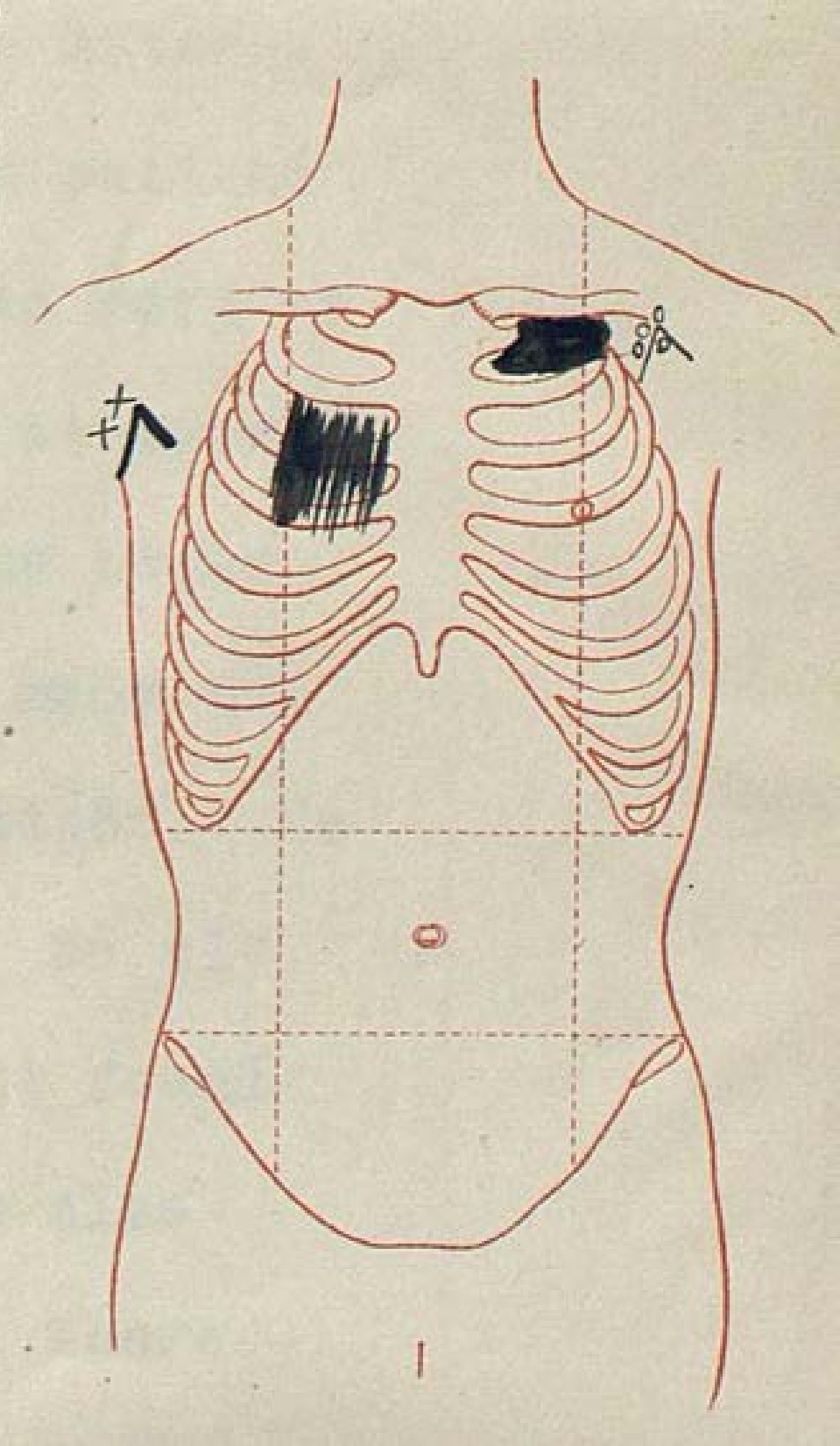
Case 1.



Had left off work when first seen. Disease must have been in existence for more than two years as he had "not felt quite well for a long time", and frequently felt giddy while at work. He had been steadily losing weight and was somewhat anaemic. For a time he seemed to improve and was able to be out for two or three hours every day. Suddenly the cough grew much worse, the dyspnoea increased and he had to take to bed. The temperature was hectic in character and the pulse rapid. There was considerable leucytosis, but no albuminuria. No tubercle bacilli could be found in the sputum. He insisted on getting up a fortnight later, and once more began to improve, though he became more and more emaciated. Diarrhoea now set in, which was with difficulty kept in check. Eventually he took finally to bed, a fairly copious haemoptysis set in and he now felt too weak to rise. The diarrhoea once more appeared, and albumen was now found in the urine. Examination of the sputum revealed numerous tubercle bacilli. He became suddenly dyspnoeic one evening and died early the following morning. The age at death was forty-one. In this case the chief complications were emphysema, diarrhoea and tuberculous invasion of the left base which extended until the whole of the lung was involved.

Case 2.

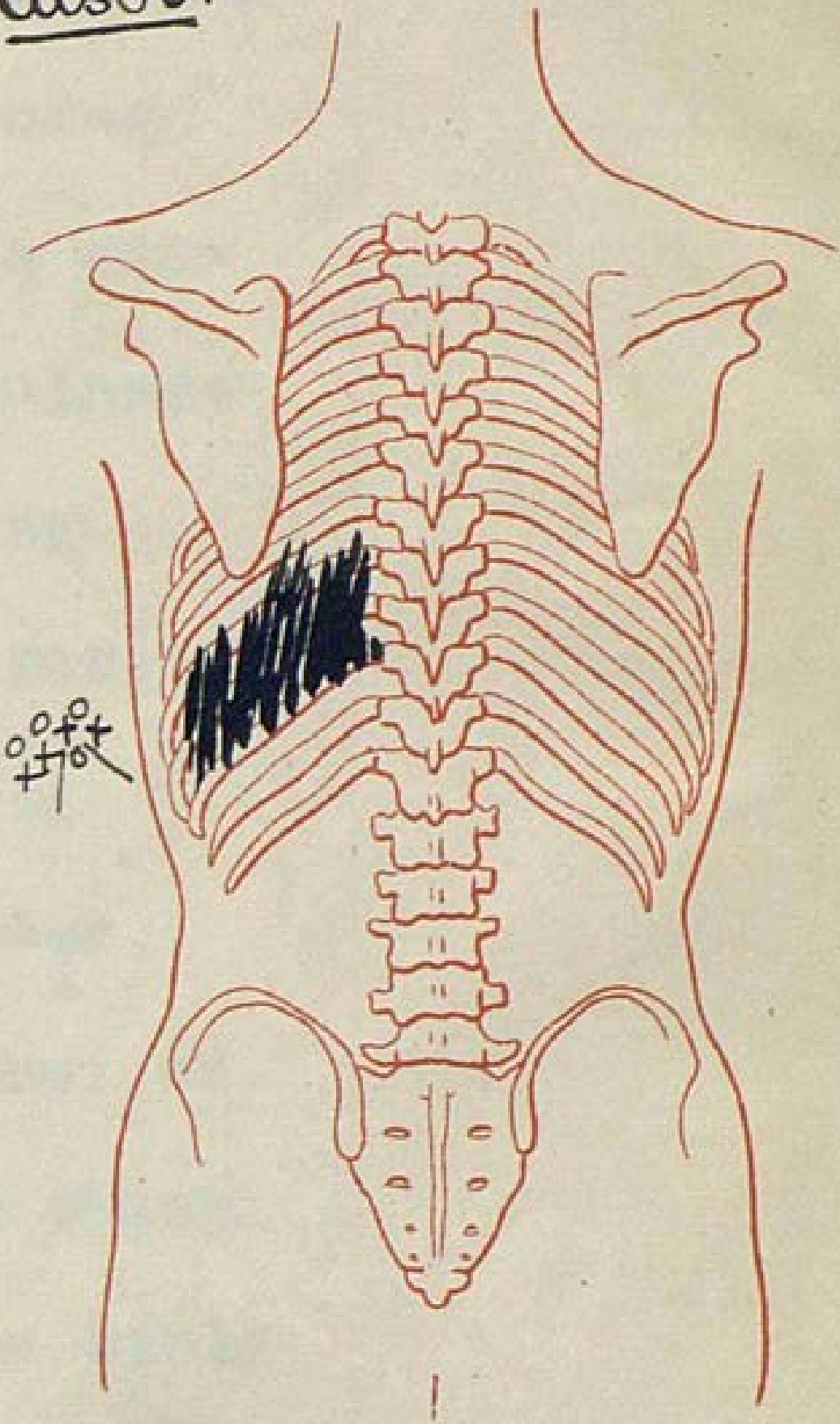




Case 2. R.L., aet. 37, married. Had been a stone- hewer for the last twelve years, prior to that he had worked as a builder and hewer at different times. Temperate. Home surroundings excellent. Both parents rheumatic. Complaint - vague pains in chest. Duration four or five weeks. Physical examination negative. Six months later had an attack of pleurisy on left side. After recovery diagram mapped out (q.v.). The patient now began to suffer from chronic cough and more or less dyspnoea. He was able to continue at work, however, for two and a half years until he began to feel too feeble. The least exertion made him very breathless, and he was now losing weight. He was anaemic, and the appetite was poor. No albuminuria present, and no tubercle bacilli found on examination of sputum. The cough was also troublesome, especially in the morning. There were now signs of emphysema as well as of bronchiectatic dilatation. The heart's action was feeble, and the apex was drawn over towards the left anterior axillary line. So far as I know this patient is still alive.

Case 3. I.T., aet. 35, married. Moderate drinker all his life. Home surroundings bad, house dark and foul smelling. Family history apparently good. Complaint "weakness and not fit for work". Small patch of dulness at left base,

Case 3.



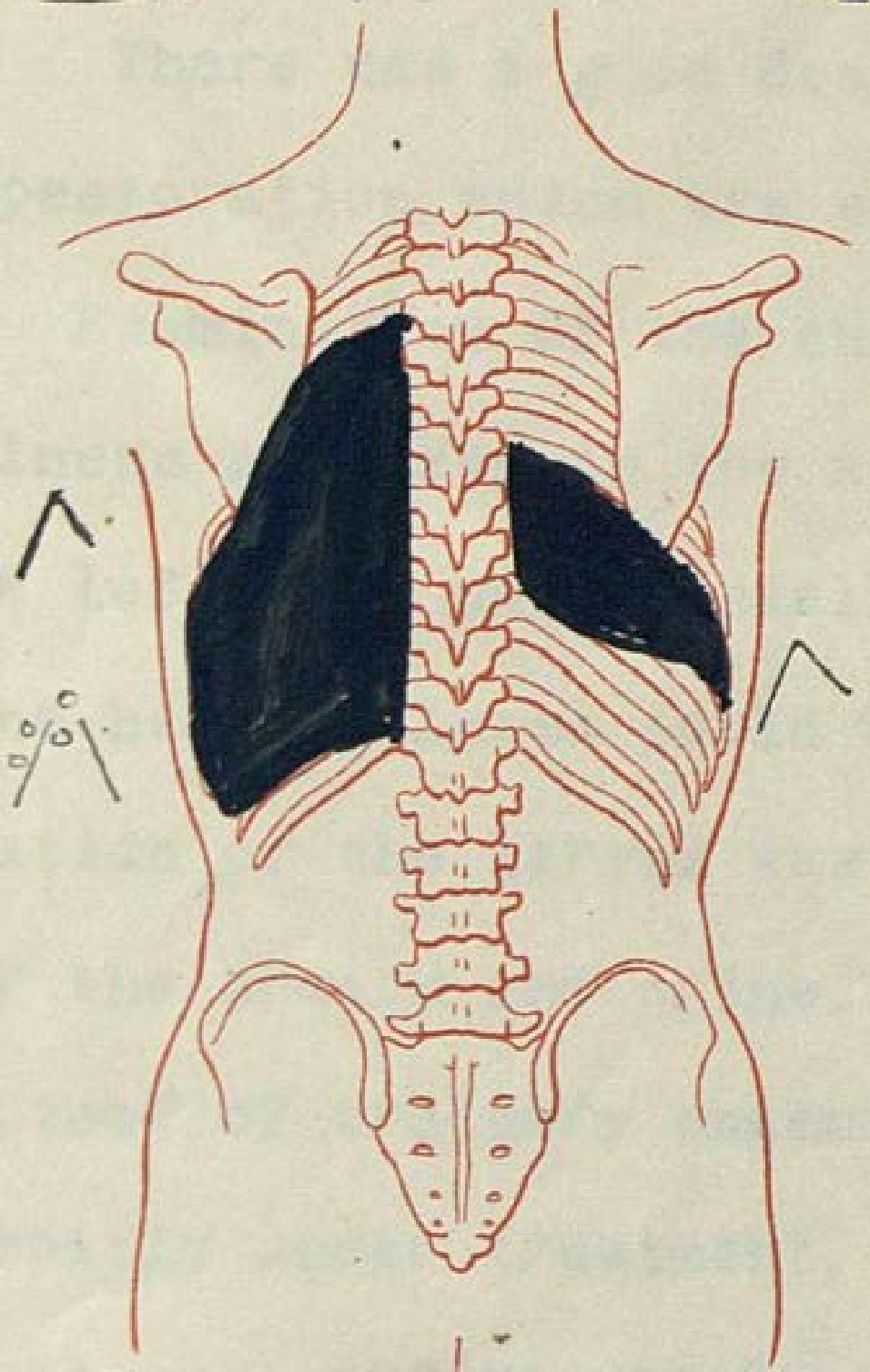
but not well marked. Had been feeling ill for some weeks. Under tonic treatment he improved and resumed work. A year later he came complaining of dyspnoea and morning cough. He had now a considerable amount of emphysema. The basal dulness was now better marked, and a peculiar creaking sound could be heard on listening over this portion of the lung. After a month's rest he resumed work, only to be laid up again six months later. On this occasion he had slight haemoptysis, but no tubercle bacilli were detected in the sputum which was very putrid and copious especially in the early part of the day. This patient has never been able to go back to work, and at present his condition remains fairly stationary, and even his weight is exactly the same (8 st. 2 lbs.) as it was nine months ago.

Case 4. R.T., aet. 36, married. Temperate. Home surroundings fair. Father died of "kidney trouble", Mother of "rheumatism and heart disease". Complaint "swelling of stomach after food and sickness". Duration three or four months. On enquiry cough was found to exist but was very slight. Patient was very constipated. Slight gastric dilatation present. Left base dull and one or two rhonchi heard on auscultation over it. Patient improved under treatment and resumed work. Next year about the same time he had a severe attack of

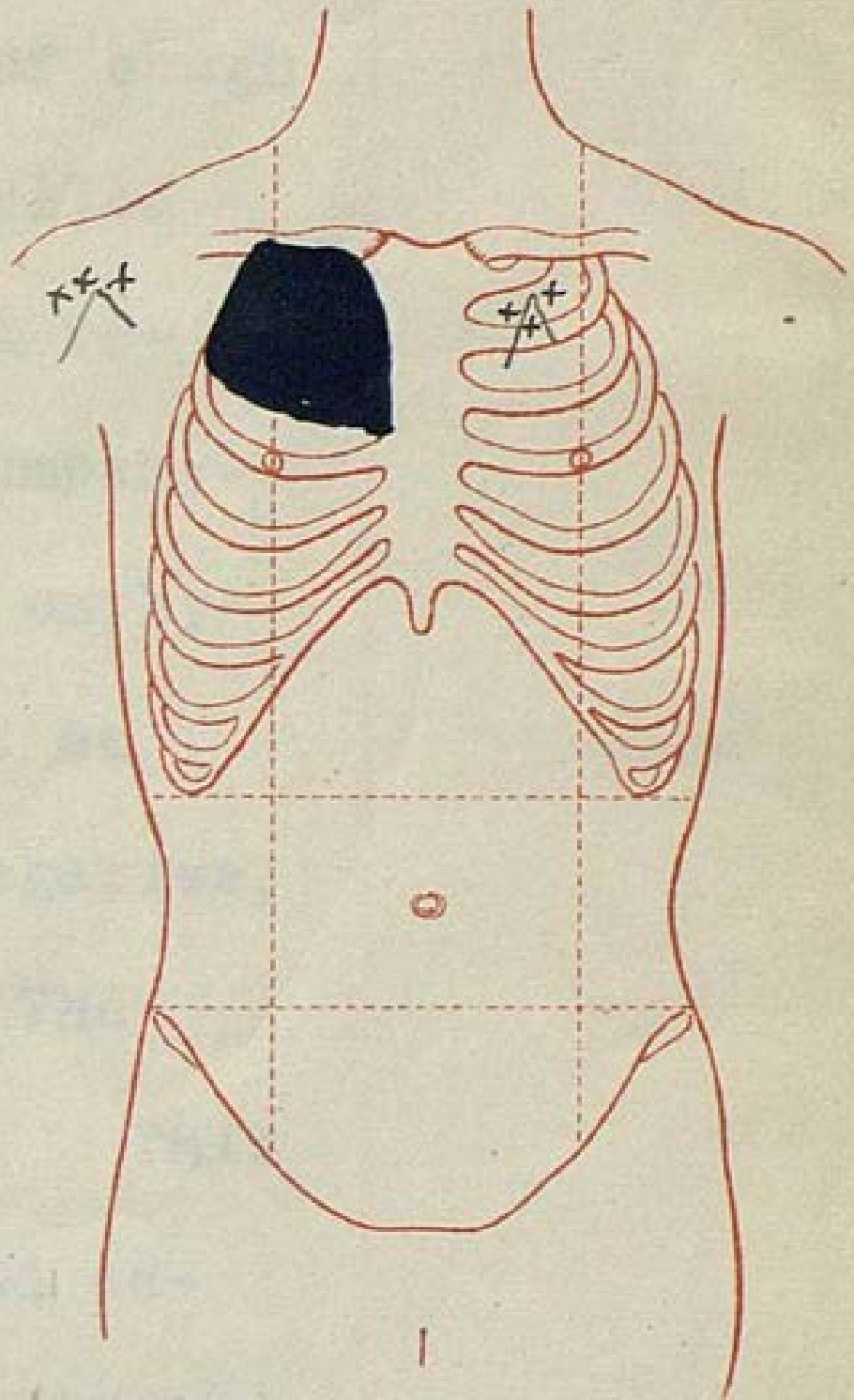
vertigo while at work, and had to go home. The chest was now found to be distinctly emphysematous and shewed unmistakable evidence of extension of the fibrosis. He improved once more; but the cough remained, while the dyspnoea became more or less constant. He was now advised to give up his work, but as this was quite out of the question he sought employment in another "yard". Evidently the conditions under which he worked were now greatly improved, for the patient's cough lessened considerably, although the dyspnoea was still very troublesome. No tubercle bacilli were ever found in the sputum, and the patient's weight has remained practically stationary for the last eighteen months. Doubtless the prognosis in this case would be fairly good, provided the patient could give up work entirely.

Case 5. J.T., aet. 40, married. Temperate, but formerly alcoholic. Home surroundings fair. Family history somewhat uncertain, but father died of some nervous trouble the symptoms of which, as described by the patient, corresponded exactly with those of paralysis agitans. Complaint shortness of breath and want of strength. Patient had been getting steadily weaker for the last two years. It was specially noted that he was extremely husky. His illness began after a wetting about two and a

Case 5.



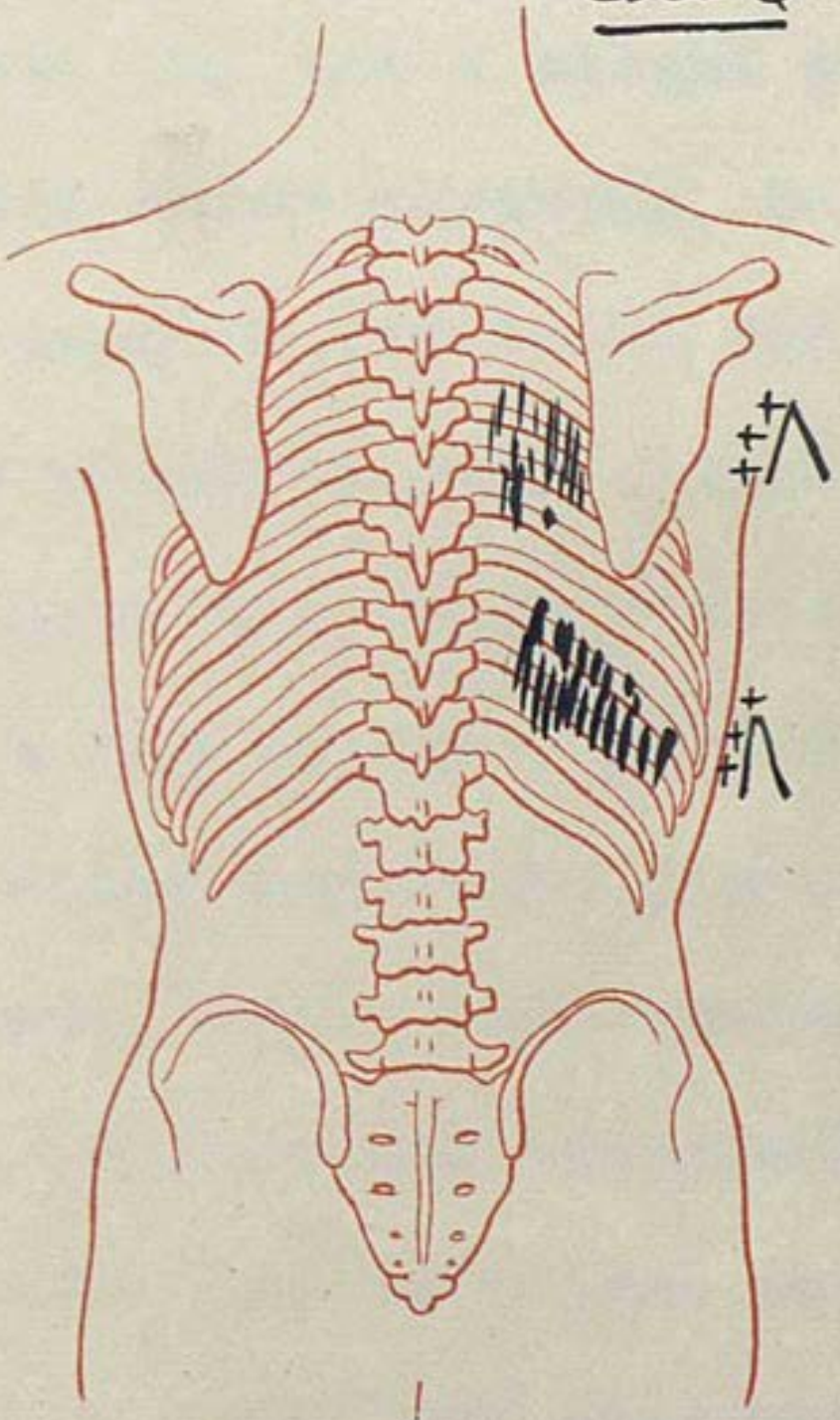
Case 5.



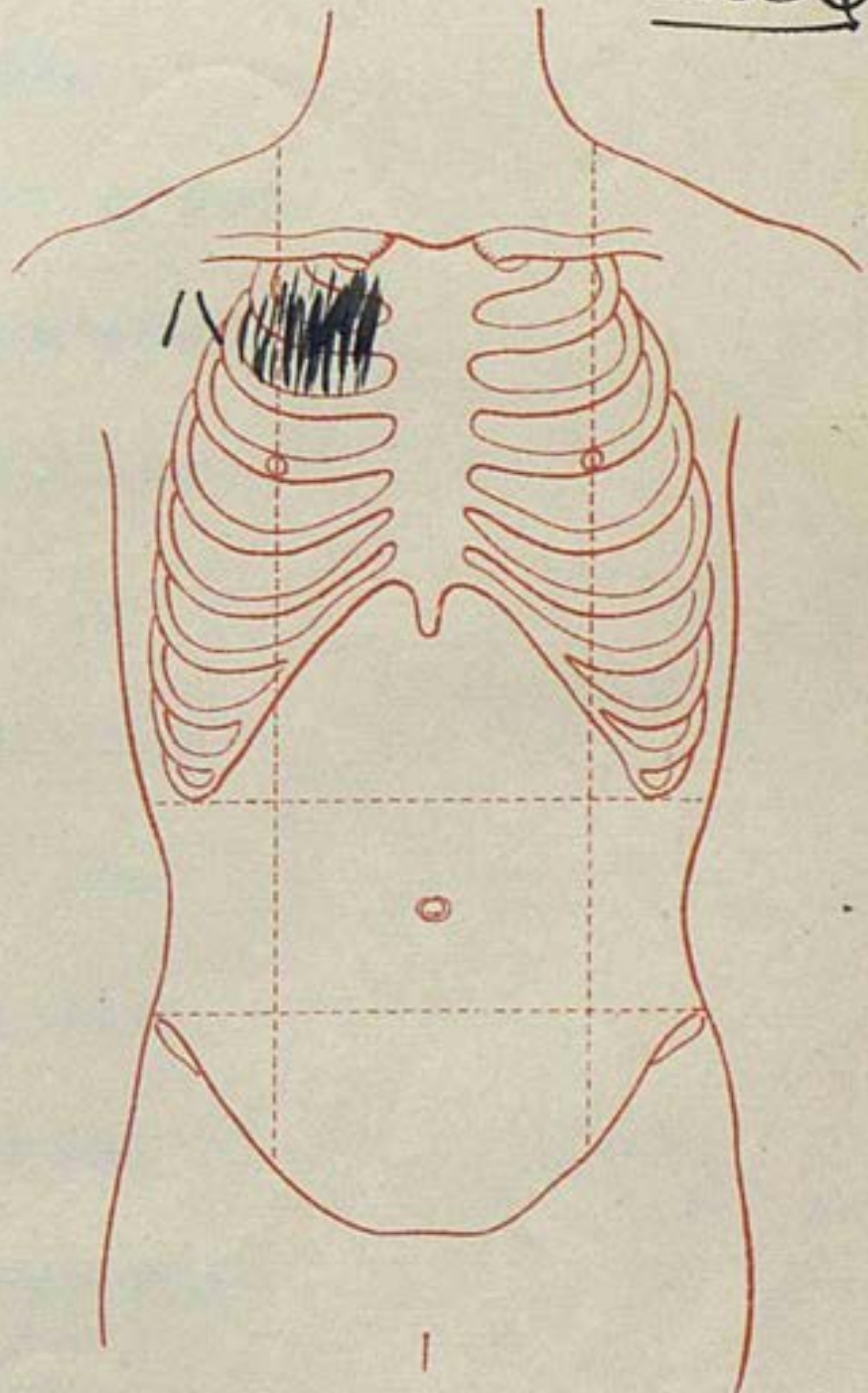
half years ago. He never felt quite the same after, and had frequently to stay off work for two or three weeks at a time on account of breathlessness and weakness. There was a good deal of cough associated with expectoration which was expelled with difficulty. Examination of the chest revealed areas of dulness as shewn in the accompanying diagrams. The left lung was specially emphysematous so that the heart dulness could not be made out. Examination of the larynx was negative, as was also that of the sputum and urine. The blood count was typical of ordinary anaemia. This patient improved greatly under treatment, and actually put on weight; but unfortunately he started work contrary to orders, and relapsed into his former condition of dyspnoea but had now a pretty severe cough. He rapidly lost weight, and signs of caseation became evident at the right apex. Tubercle bacilli appeared in the sputum, the blood count shewed slight leucocytosis and the temperature became swinging in character. Slight attacks of haemoptysis occurred from time to time. He now rapidly went downhill and the ankles began to swell, while the urine contained a considerable quantity of albumen. He died of heart failure at the age of 42, exactly two years after I first saw him.

Case 6. D.C., aet. 42, married. Temperate.

Case 6.

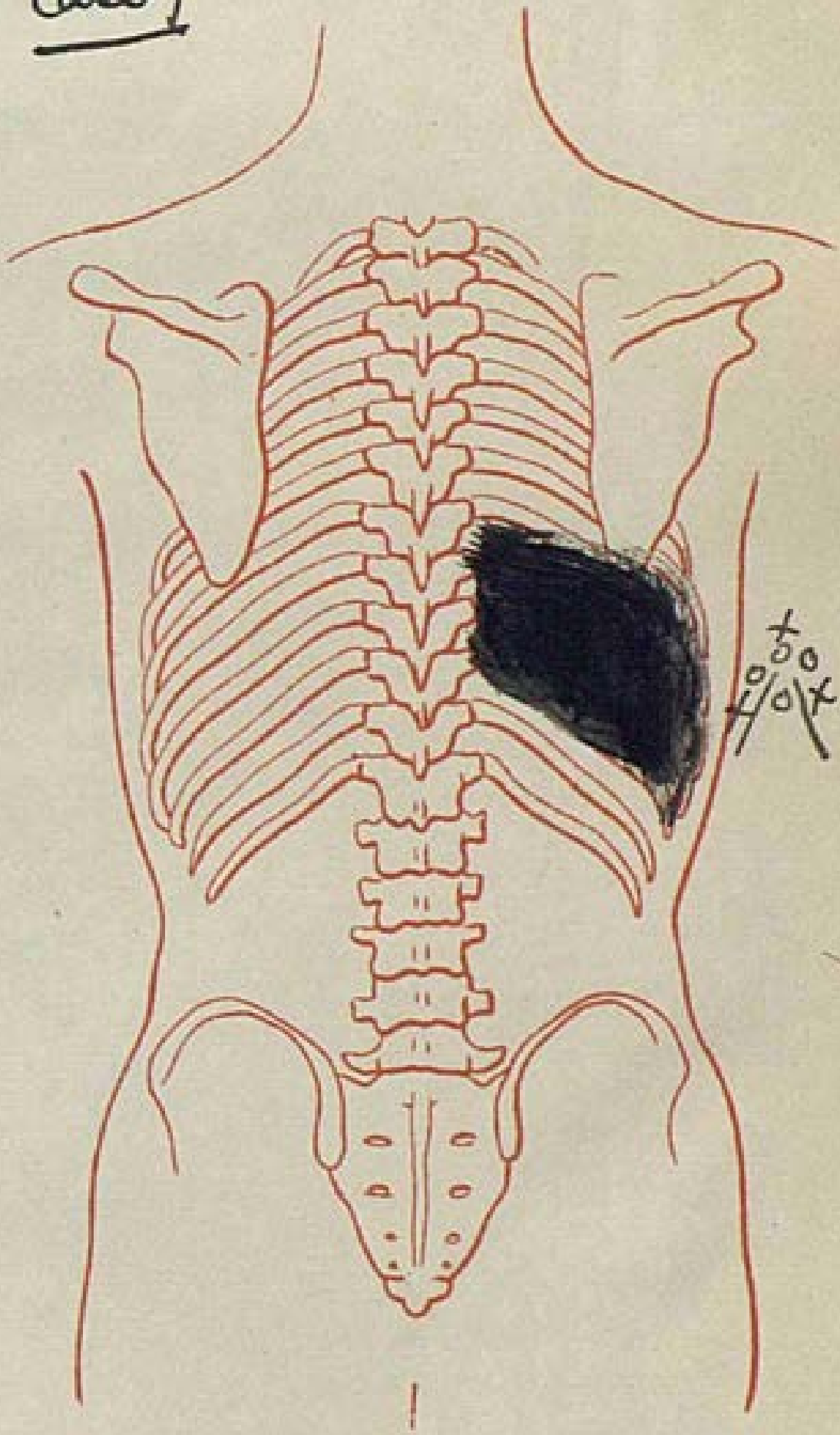


Case 6.



Nothing known as to home surroundings. Father rheumatic, mother strong and healthy. Had consulted me four and a half years previously for gastric disturbance. He was in the habit of "carrying" all his food, and had an uncomfortable loaded sensation after every meal. He was also very much troubled with constipation. There was no cough or dyspnoea and no heart trouble. Examination of the urine revealed no abnormal condition. Under treatment he soon recovered. He continued to see me at various times in the interval when his complaints varied from dyspepsia to a feeling of tightness in the chest and pains in the right side. Examination at no time indicated any pulmonary change. Recently, however, he came complaining of a "severe pain just under the right shoulder blade". He was also breathless and had a slight cough. There was considerable malar flushing, but no great loss of flesh. I examined the chest very carefully behind and found two areas of very slight dulness over which the breathing was harsh, vesicular in type accompanied by a few rhonchi, chiefly during inspiration. Just below the right clavicle the dulness was also marked, while the breathing was distinctly bronchial without any accompaniments. The percussion note over the left lung was somewhat hyper-resonant, and the heart was considerably

Case 1.



dilated. The pulse was markedly irregular. The patient said that if he could get rid of the pain he would be all right. I have no doubt that in this case the fibrosis, too slight in amount to be detected before, is now advancing and that unless this patient gives up his work altogether the condition will progress in the usual way.

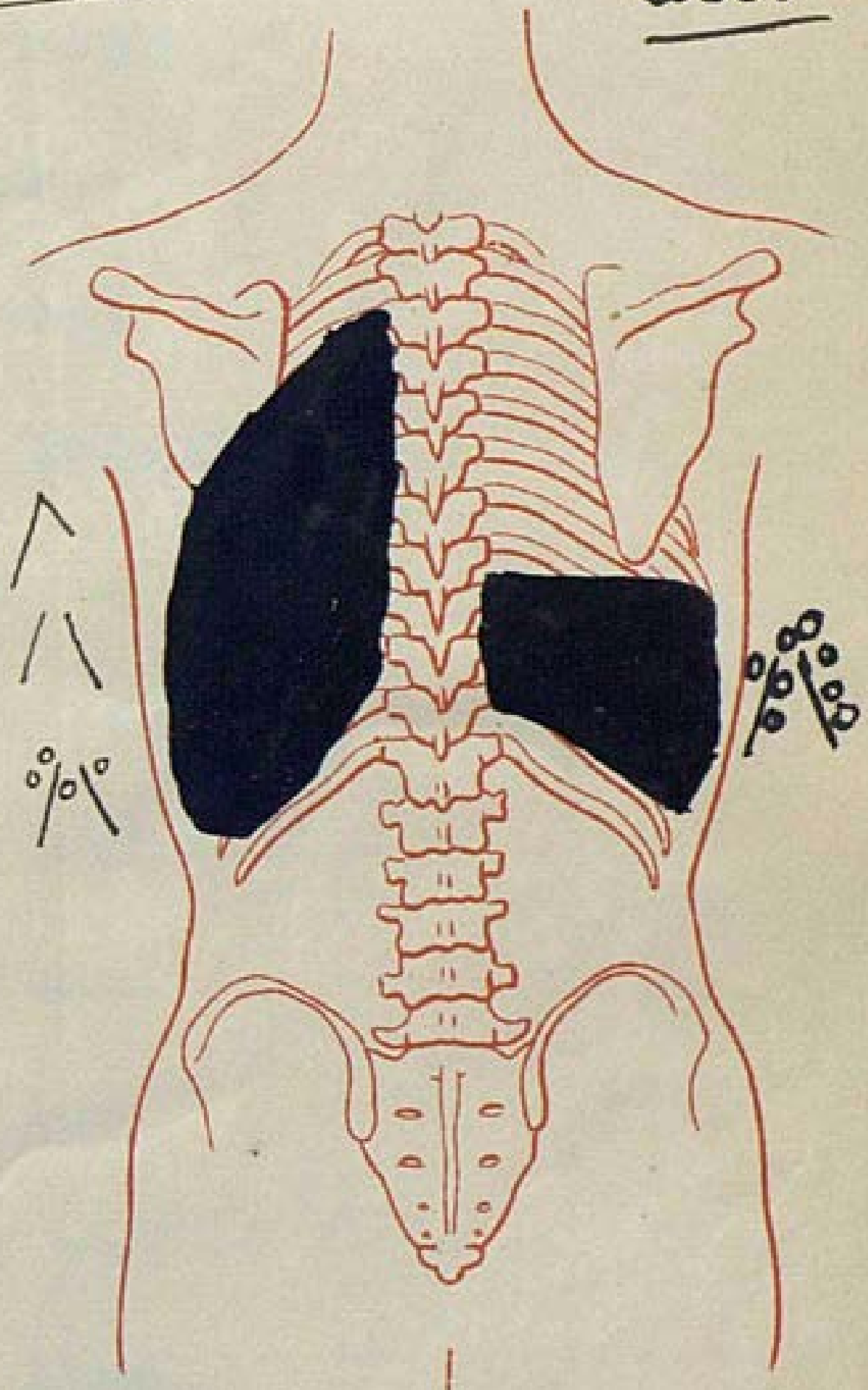
The next two cases are particularly interesting, as illustrating well how treatment and cessation from work may prolong life considerably even under the most unpromising circumstances.

Case 7. J.P., aet. 36, married. Had never been very temperate. Father rheumatic; mother died of gastric carcinoma. Home surroundings most unhygienic. House consisted of a room and kitchen which accommodated six adults and three children. Food was abundant, but indifferently cooked. Illness began somewhat suddenly with haemoptysis. Patient had, however, been complaining of cough for some weeks before, but had paid little attention to it. At my first visit he was sitting up in bed. The pulse was rapid and the respirations numbered thirty per minute. The temperature was normal. On examination of the chest distinct dulness was obtained over the right base associated with numerous rhonchi and crepitations. The apices were free. No tubercle

bacilli were found in the sputum. The amount of blood coughed up was considerable, although I had no means of determining the exact quantity, as the patient expectorated into a chamber-utensil which was half filled with urine. The room was exceedingly filthy and smelt horribly, while the bed-clothes were soiled beyond description. Under treatment, however, this patient made a perfectly good recovery, and only the dulness remained behind. He was advised to seek some other occupation, but being a naturally lazy man he preferred to live on the earnings of his family. For two years he remained in comparatively good health, and then one day he got wet while out walking. He now had a severe bronchitic attack, and when this cleared up the patient began to suffer from breathlessness and vertigo. He also lost a good deal of weight and became very anaemic. The fibrosis now extended and involved the whole of the right lower lobe and part of the left. Since then the cough has never left him and he now expectorates a good deal of material especially in the early part of the day. He has marked emphysema as well as bronchiectasis, but although it is five years since he had the attack of haemoptysis the patient is able to go out every day, and can walk a good distance. There are no tubercle bacilli in the sputum, and his weight has remained practically stationary for the

Pleuren.

Case 8

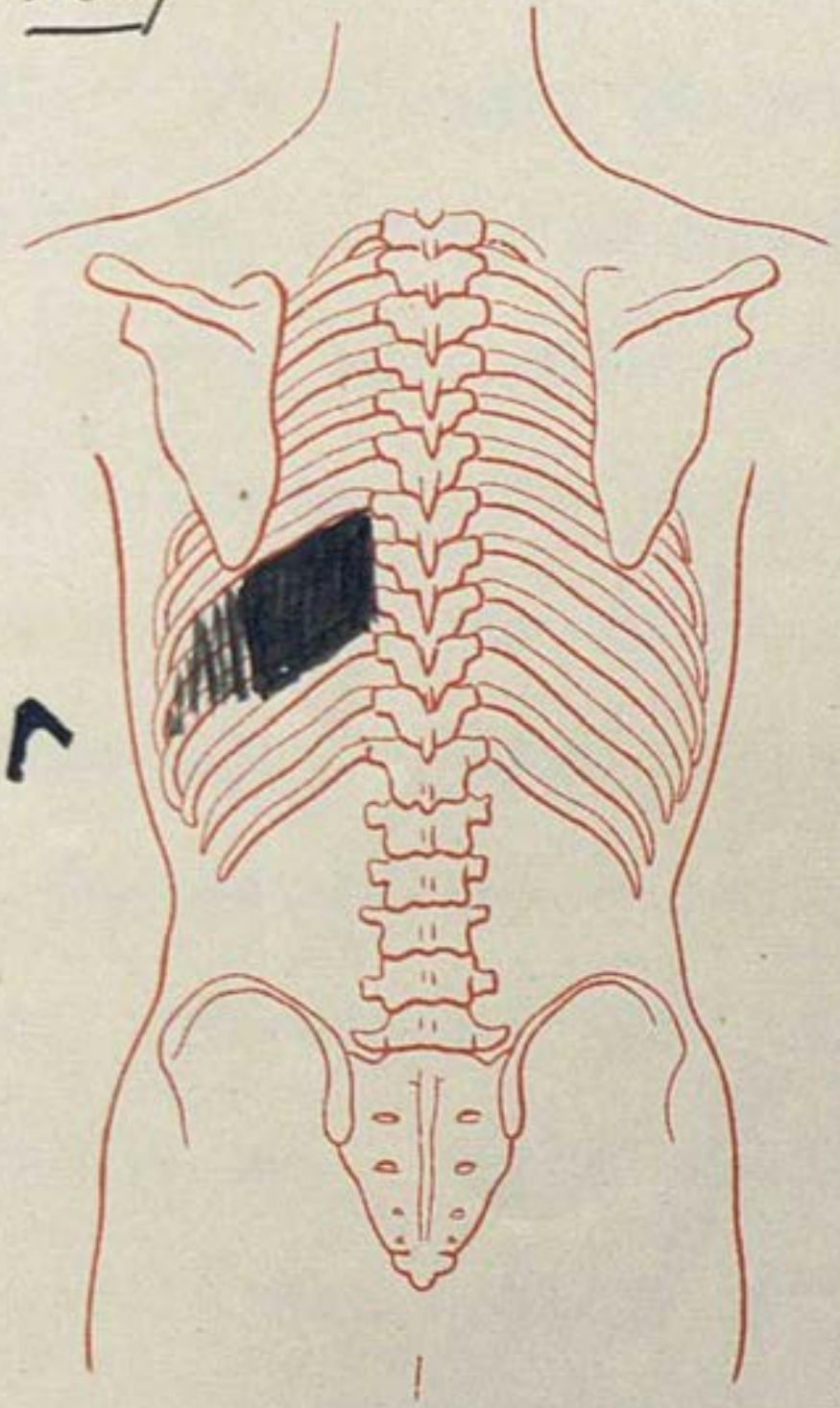


last two years. He takes his food well, and sleeps well. He has occasional attacks of slight indigestion, probably due to his eating unsuitable food. To all appearnace this patient is likely to live for a good many years yet, although his surroundings are anything but favorable.

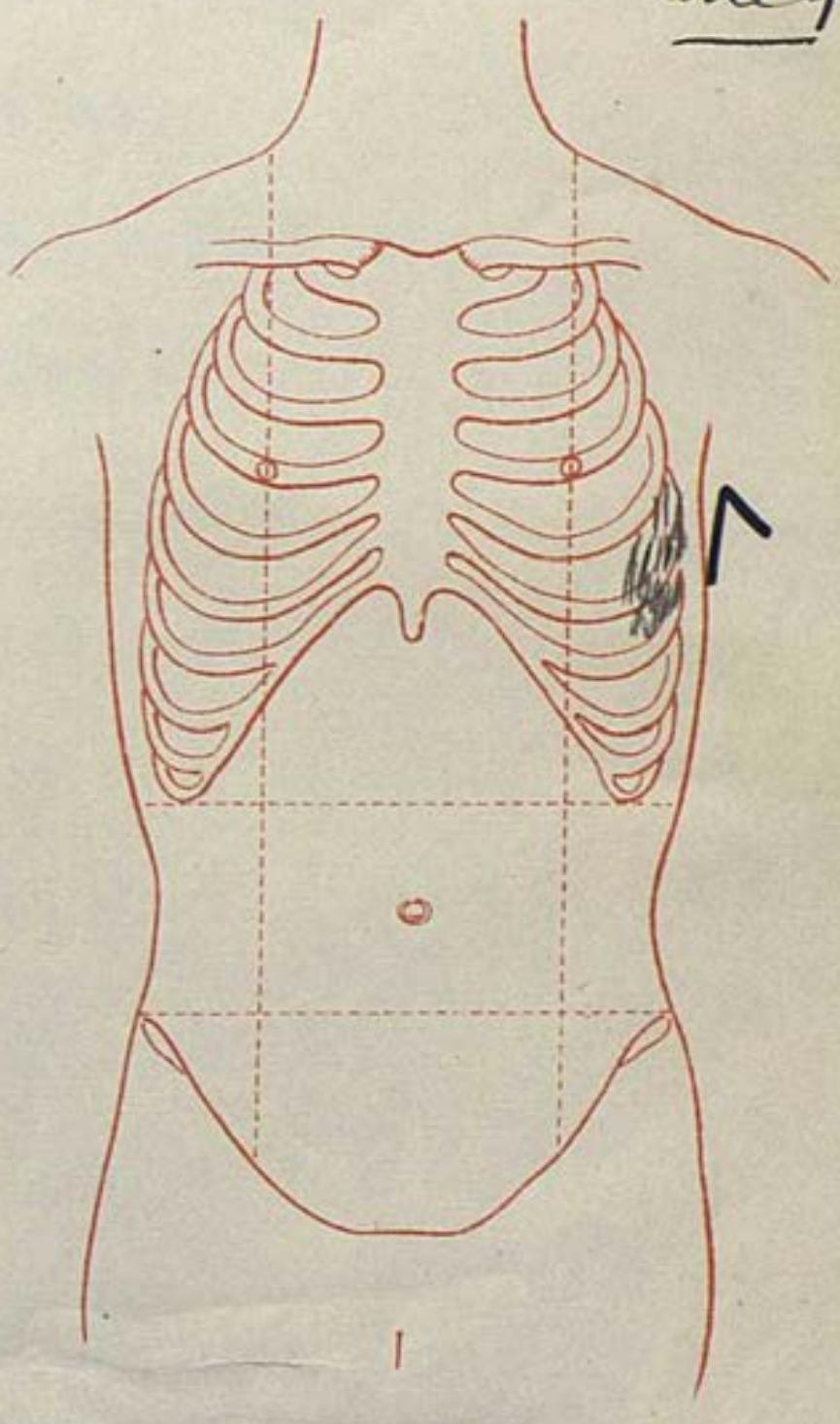
Case 8. D.M^{c}L., aet. 37, married. "Always drinking whenever he gets the chance" (wife's statement). Home surroundings excellent, as his wife believed in the value of fresh air and good food. Parents both quite healthy. Had always been quite strong and well until two years ago when he had an attack of pneumonia from which he made a very slow recovery. Had always suffered from cough and more or less expectoration ever since. Was breathless at times. His present complaint was "weakness and dyspnoea." Both lungs were found affected by fibrosis as indicated in the diagram. Over both distinctly characteristic creakings were heard on auscultation. The urine contained a trace of albumen. The blood count shewed distinct anaemia. The heart was pulled over to the right side so that the apex beat was located fully an inch inside the mammillary line. There was considerable emphysema present as well. Examination of the sputum was negative. Patient remained off work for six weeks and then resumed it.

He remained in much the same condition, now better perhaps and then again somewhat worse, for the next three and a half years. He then had a febrile attack which passed off in nine or ten days, and after which the area of dulness was found to be more extensive. There were now also unmistakable signs of bronchiectasis, and the sputum for the first time became tinged with blood. The dyspnoea was markedly increased and the pulse much feebler in character. Rest and careful treatment, however, again enabled the patient to return to work; and he was able to continue at it with periods of rest for two years longer. Then he had finally to give in as he had by this time become intensely dyspnoeic. The sputum now shewed a few tubercle bacilli, and crepitations were abundant at both apices. He went about in this condition for ~~about~~ nearly six weeks, and then took to bed. Swallowing now became difficult. The cheeks were flushed, the pulse rapid, the breathing hurried and difficult, and the lips markedly cyanosed. The ankles and legs began to swell considerably. The blood shewed very well marked leucocytosis, while the urine contained a little albumen. The day before his death the patient became suddenly and extremely dyspnoeic. Pneumothorax was diagnosed, and he sank rapidly, being unable to utter a word or even to sip water,

Case 4



Case 4.



although he was evidently very thirsty. This patient, it will be observed, lived for a period of between seven and eight years after the first onset of the disease.

Case 9. R.T., aet. 37, married. Home surroundings fair. Both parents alive and healthy. Had always been in the enjoyment of good health until the present attack came on. He had been perfectly well in the morning, but as the afternoon wore on he suddenly experienced a sensation as if someone were trying to suffocate him. He had to leave work and go home. He went straight to bed and sent for me. When I got to the house I found him sitting up in bed, breathing quickly and evidently in great distress. He thought he was dying. His temperature was normal, but his pulse was rapid, small and feeble. At the left base there was a considerable area of dulness which became much less marked in the axillary region and faded off as the anterior axillary line was crossed. Over this portion of the lung the breathing was of a very harsh vesicular type. The heart was somewhat enlarged, but not markedly so. This patient had never complained of his chest before, nor had he even then any cough or expectoration. He was merely dyspnoeic. Rest in bed for three days afforded him complete relief. He was advised to

take a holiday; and he did so, remaining off work for a fortnight. He then resumed his employment. Nothing further occurred until nearly nine months later when he felt suddenly giddy on getting out of bed one morning. He tried to go out, but could not, and accordingly sent for me. I found that the dulness had extended, and that most of the right lung was now emphysematous, so that the right border of the heart could not be percussed. He was kept in bed, and during this period I took the opportunity thus afforded of examining his urine every second day for a fortnight.

The results obtained are interesting as well as instructive for they shew a decrease in the amount of phosphates. I have already referred incidentally to this case in speaking of the general condition of the urine in pulmonary fibrosis. In this patient's case the sulphates shewed a slight increase on every occasion on which they were estimated. I shall now tabulate the results of my quantitative estimation of the daily output of phosphates and of urea in this particular case.

Day.	Amount of Urine passed during 24 hours.	Amount of Phosphoric Acid excreted.	Amount of Urea eliminated.
1	40 ounces.	20.75	280 grains.
3	43 "	24.25	284 "

Day.	Amount of Urine passed during 24 hours.	Amount of Phosphoric Acid excreted.	Amount of Urea eliminated.
5	43 ounces.	21.50	288 grains.
7	47 "	24.00	300 "
9	35 "	24.50	292 ""
11	46 "	26.50	320 "
13	50 "	22.00	310 "
15	48 "	27.25	334 "

(Estimation of urea made by Hufner's method and the Southall-Doremus tube employed throughout. The phosphoric acid estimation was made by Neubauer's method.)

These figures are of course given in round numbers for the sake of simplicity. Generally speaking it will be observed that the diminution in urea elimination corresponded with the diminution in the phosphoric acid excretion. I was interested in observing that the phosphoric acid output was most diminished on those days when the patient was most dyspnoeic; not so, however, the urea. Phosphoric acid is usually diminished in febrile conditions, but in this patient's case the temperature was absolutely normal throughout the period during which my investigations were conducted, while the diet was but little altered, and contained a fair proportion of butchers' meat, vegetables, and farinaceous food. I am not

prepared to state what was precisely the cause of this low amount of phosphoric acid and of urea excretion in this particular case; although probably some more or less definite tissue change accounted for it, but what the precise nature of this was I am of course unable to say.

After the patient's recovery he again resumed work, but was never quite the same again. He now began to complain of cough with some expectoration, while at times his dyspnoea became very troublesome. Eventually he had to give up work altogether. This was fully three years after the onset of his first symptoms. The condition of the chest was now somewhat as follows:- The left lung was more or less dull throughout and definite "creaking sounds" could be held at various places, especially in the axillary region. The upper part of the right lung was emphysematous, but towards the angle of the scapula the physical signs of a large bronchiectatic cavity could be easily made out. The patient now began to lose flesh more rapidly; and at this time I again examined the urine every alternate day for six weeks, and the blood once a week during the same period. The following table gives in round numbers the results of the urine and blood estimations.

[TABLE.

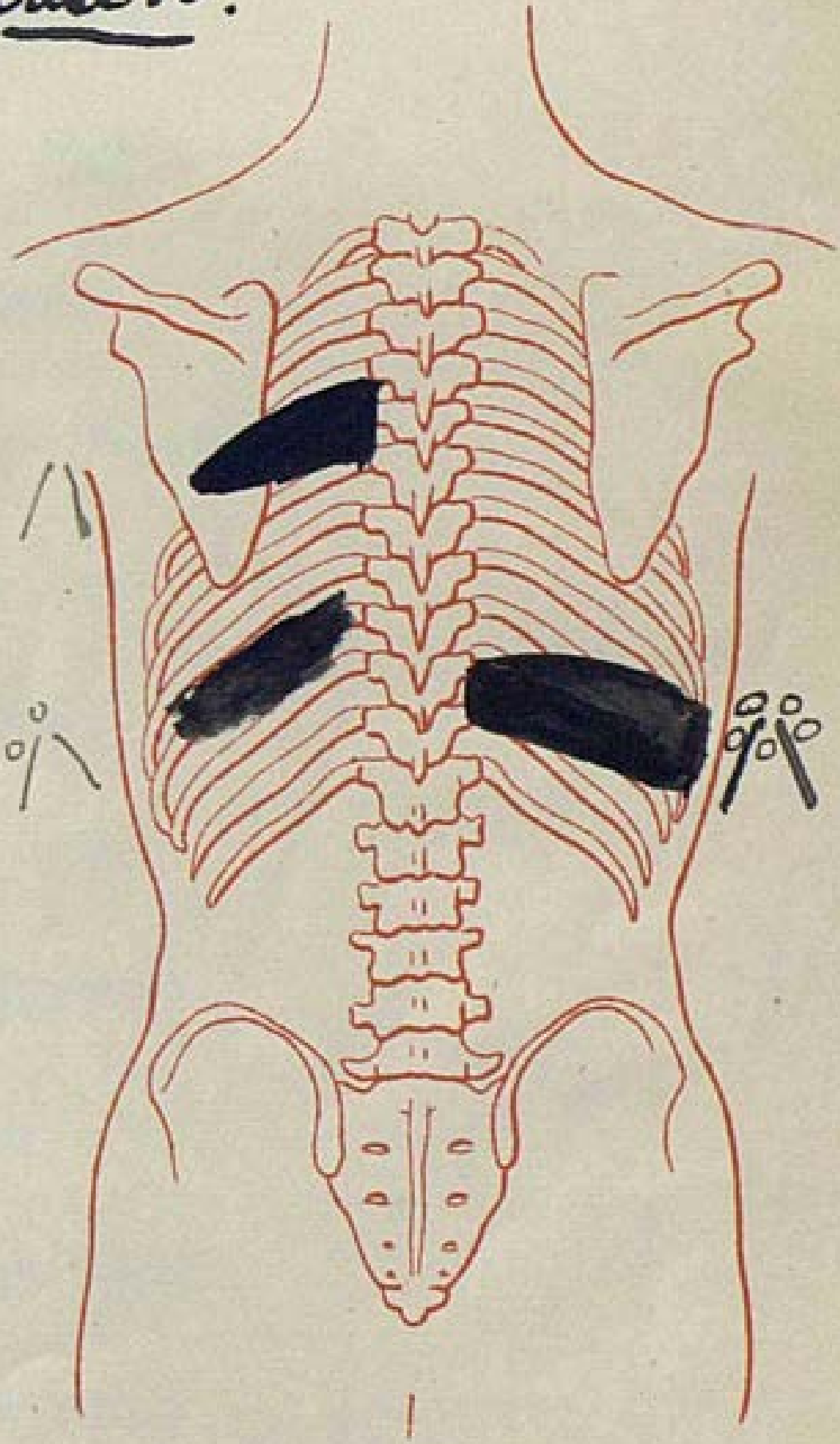
Day of Observation.	Percentage of Haemoglobin.	No. of R.B.C.	No. of Leucocytes.
1	50	4,000,000	4,850
3			
5			
7			
9	51	4,200,000	4,750
11			
13			
15			
18	54	4,200,000	4,900
20			
22			
24			
26	52	4,050,000	5,000
28			
30			
32			
34	52	3,880,000	4,800
37			
39			
41			
43	51	4,000,000	4,600

Amount of Urine passed.	Amount of Phosphoric Acid.	Amount of Urea.
43 ounces.	20 grains.	290 grains.
47 "	22 "	286 "
49 "	22 "	288 "
38 "	20 "	292 "
38 "	24 "	310 "
41 "	26 "	314 "
40 "	21 "	300 "
50 "	24 "	320 "
48 "	24 "	340 "
46 "	20 "	300 "
38 "	21 "	320 "
40 "	24 "	360 "
43 "	23 "	325 "
42 "	24 "	236 "
46 "	26 "	350 "
49 "	25 "	289 "
40 "	21 "	295 "
46 "	21 "	290 "
39 "	22 "	306 "
38 "	21 "	275 "
43 "	25 "	318 "

The blood examinations throughout shewed great diminution in the haemoglobin percentage, with decrease of the erythrocyte count and also of the leucocytes, while the red blood corpuscles bore evidence of well marked poikilocytosis. The urea was diminished, as was also the phosphoric acid, although on some days the decrease in the former did not correspond to a proportionate decrease in the latter. On the whole, however, the output of phosphoric acid and of urea were relatively diminished and proportionately so throughout the series of examinations which I made. The sputum contained no tubercle bacilli.

The disease now progressed steadily, and at the left apex numerous crepitations could be heard, while the breathing was distinctly bronchial in type. The cough and expectoration became more frequent until the patient was never free from these troublesome symptoms. Examination of the sputum revealed a fewtubercle bacilli, but the urine remained free from albumen. This was the patient's condition five weeks before death took place. Crepitations could now be heard on both sides of the chest, and there was a marked cavity at the left apex over which the breathing was cavernous in character. The patient now took to bed entirely and became a mere skeleton. The

Case 10.



cheeks were flushed, while the lips and nose were purplish in colour. He gradually grew worse and worse until he eventually died quite suddenly while reaching out his hand for a cup which he used as a spitoon. A week before death there was evidence of leucocytosis and the urine contained a trace of albumen. The ankles were also somewhat puffy, and the patient had a slight attack of diarrhoea which lasted for three days.

I have thought it advisable to refer to this case at greater length as it was one in which I carried out several clinical observations, not always so easy to make in private as in hospital practice.

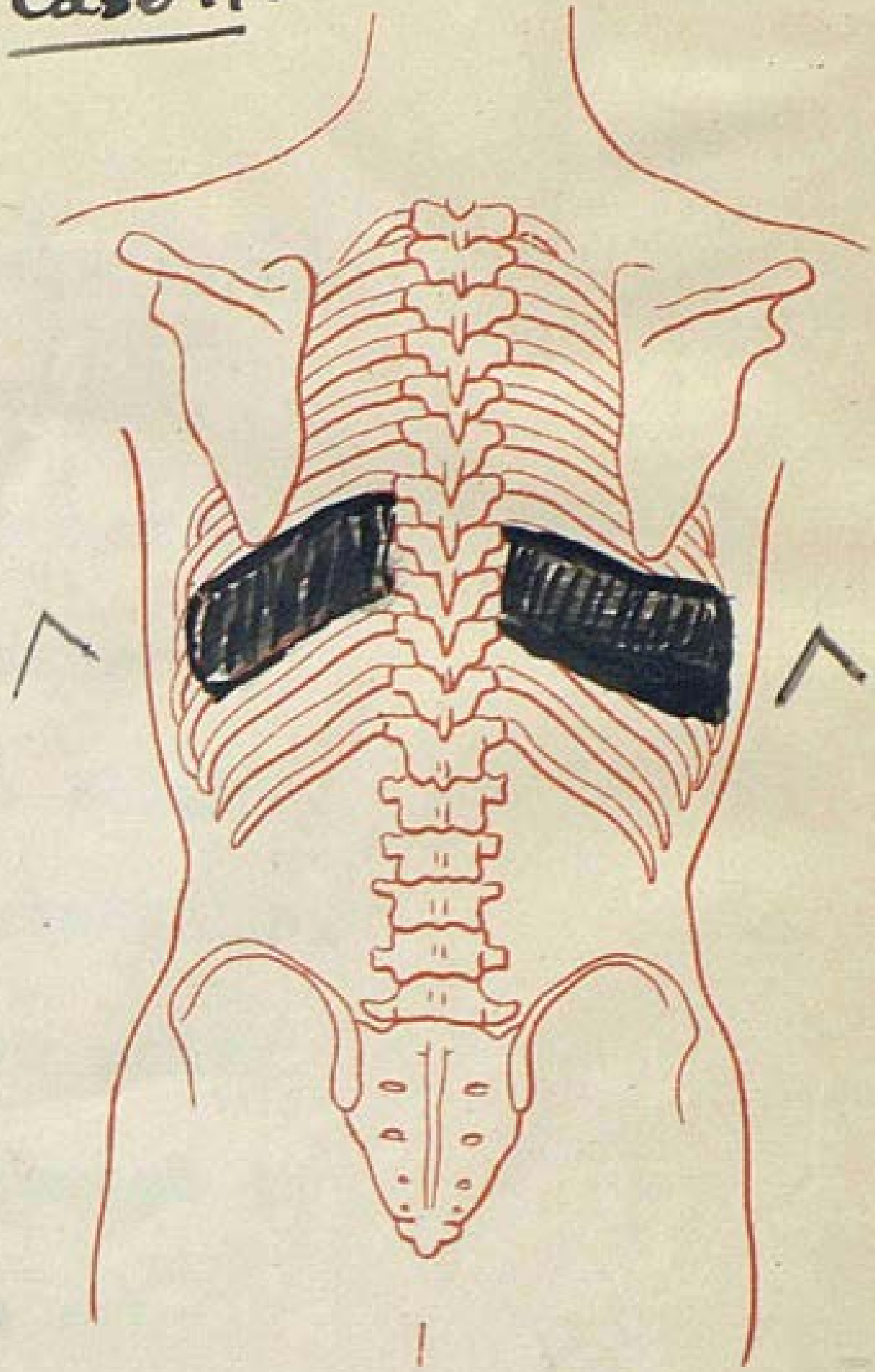
Case 10. R.W., aet. 38, married. Home comfortable. Very alcoholic at one time, now a moderate drinker. Father dead, suffered greatly from rheumatism; mother died in childbed. Complained of cough and breathlessness. Had been ailing for fully two years. Illness said to have begun with an attack somewhat like influenza in its symptoms. Patient thin and pale with distinct clubbing of fingers. Dulness at both bases and also in left scapular region. Right base shewed very marked bronchial breathing with numerous coarse crepitations. At left base breathing was also bronchial, but not so harsh, and crepitations

were less numerous and not so bubbling in character. Patient could only do a little light work, but absolutely unable to work for more than three or four hours a day. No tubercle bacilli in sputum. Blood examination shewed reduction in all the elements. This patient was under my care more or less constantly for the next two years, being sometimes better and sometimes worse, but always able to go about. Three years ago his wife, who had been working hard, doing washing and cleaning, rising early in the morning and going late to bed, began to have a hacking cough and one day she spat up a little blood. At her left apex evidence of caseation was already present. She took to bed and in less than six weeks died of general tuberculosis. Her sputum was loaded with tubercle bacilli. As she slept with her husband it was thought that he had infected her. At no time was I able to detect the presence of organisms in his sputum, but it is quite possible that he may have had a tuberculous change of a somewhat chronic character grafted on to the fibrous disease of the lungs. This case is instructive as illustrating the possibility of a tuberculous lesion being present in those cases although the examination of the sputum is altogether negative. Of course this is going on the supposition that the wife in this case

caught the infection from her husband. The latter is still alive, and sees me occasionally. He is now much thinner; but beyond slight breathlessness he complains of little, and is out of doors for a long period each day. His cough is very slight, but his chest is markedly emphysematous. There is little or no extension of the dulness over the lower parts of the lungs. He has had a troublesome hydrocele which has now been tapped three times. There are still no tubercle bacilli to be found in his sputum, nor albumen in his urine, and he eats and sleeps well. In fact he looks as if he might live for another ten years, although his age is now about forty-three.

Case 11. J.S., aet. 37, unmarried. Lives in a very comfortable house. Consulted me on account of fainting attacks which he had had the week before. While in a friend's house on the Thursday night he felt faint, and had to lie down. He was unconscious for about five minutes. Next day the same thing occurred while patient was at work. He could not account for them as he had always been healthy hitherto. He had been a builder for a number of years, but recently owing to dull trade he had to take to hewing. He was not a teetitaler, but he never drank to excess. Right side of the heart was slightly enlarged. At both bases a

Case II.

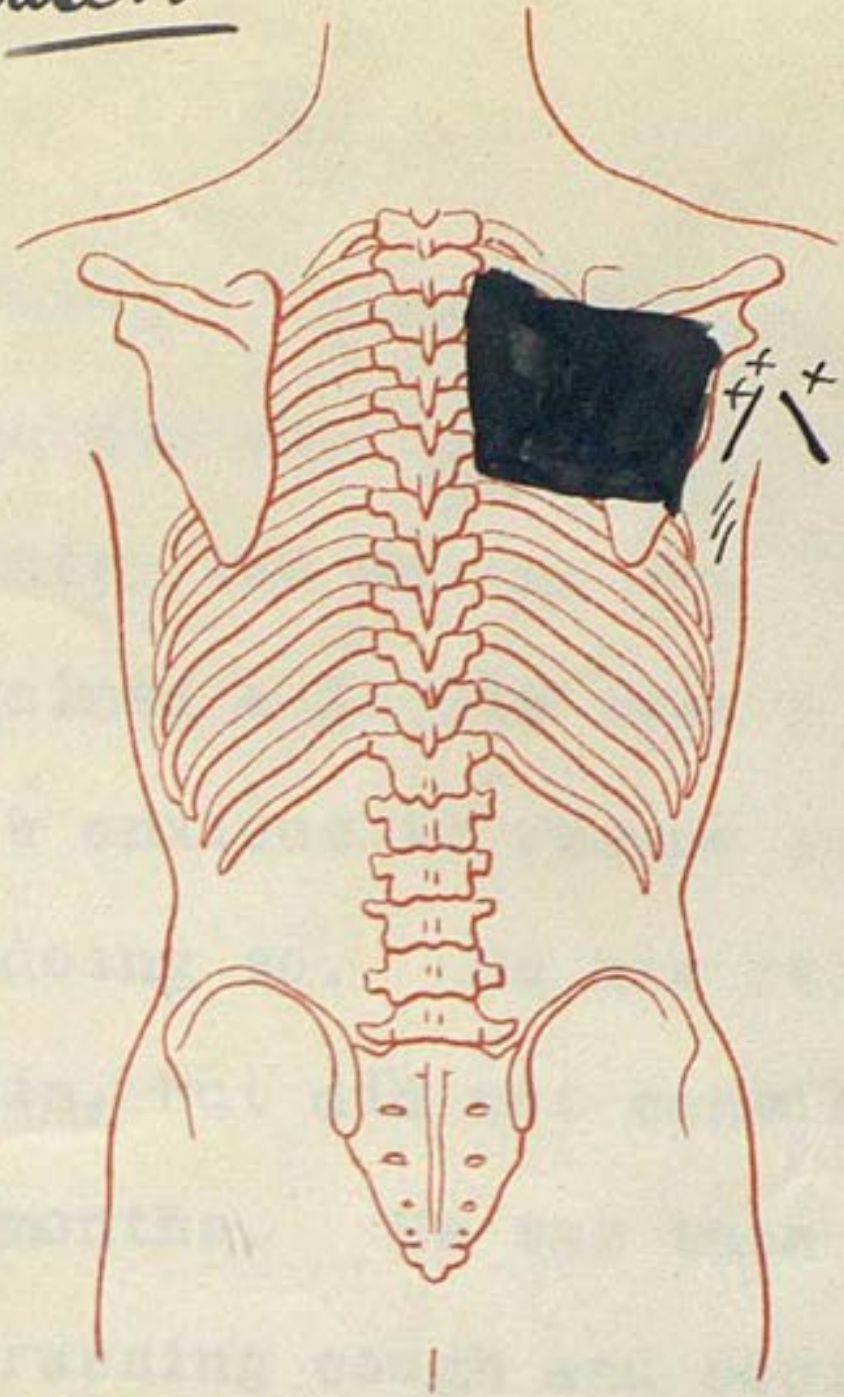


certain amount of dulness, but by no means absolute, was made out; and over these areas the breathing, especially on the right side, was distinctly harsh vesicular in type. On being questioned further the patient admitted that he sometimes had a slight cough in the mornings, but without any expectoration, though he was not breathless. He was placed under treatment, but has not since returned; although I am told that he has had no recurrence of the syncopal attacks and is at his work every day.

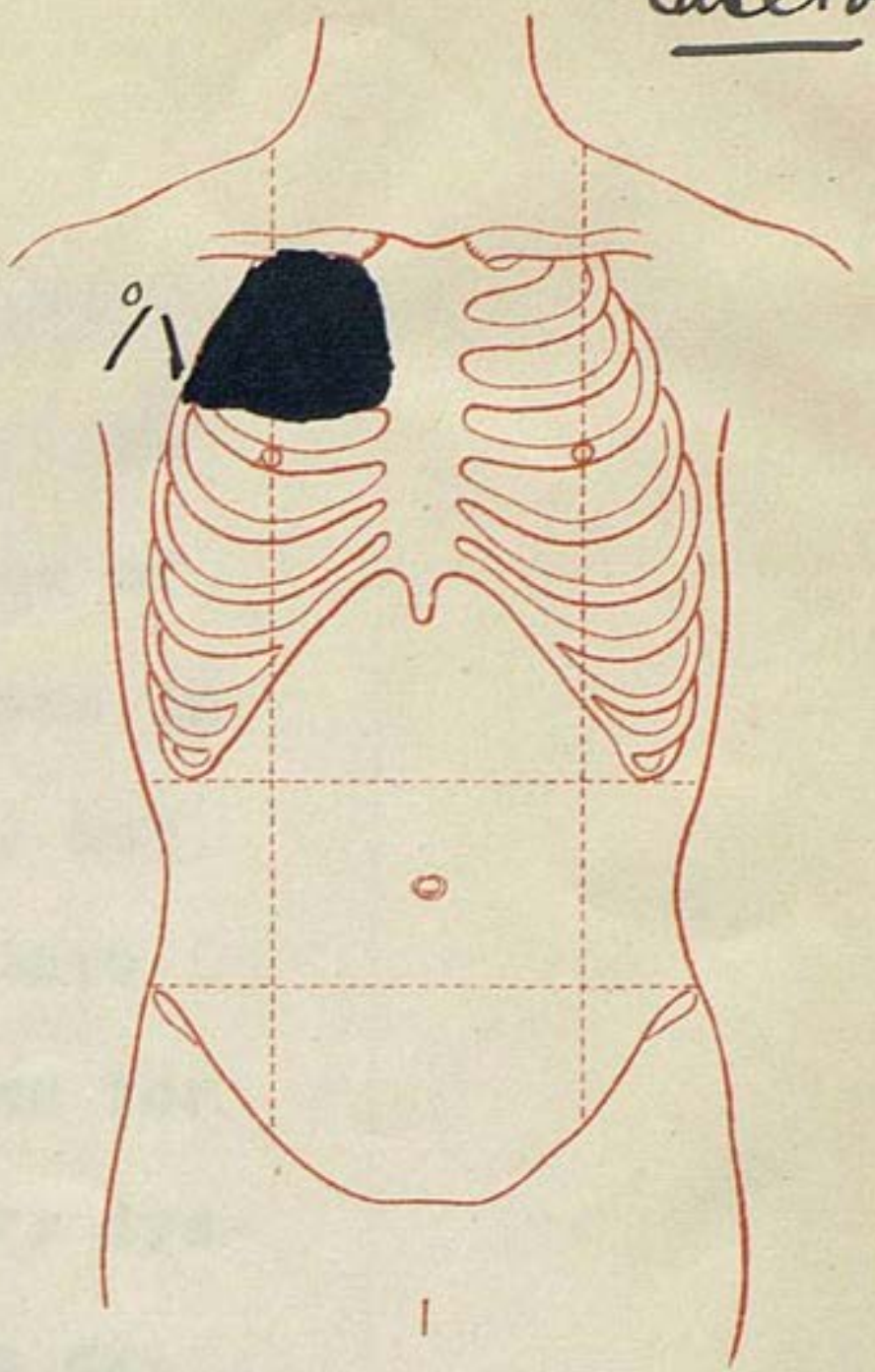
It will be observed that in this patient's case the condition for which he consulted me did not suggest pulmonary disease, and unless I had insisted on making a thorough examination of the chest the true nature of his illness would have been altogether missed. This case, therefore, well illustrates the importance of being thorough in our examination of every patient presenting himself for treatment.

Case 12. J.P., aet. 33, married. Home comfortable. Alcoholic. Complained of sickness after food and giddiness. No cough and no dyspnoea. Improved under treatment. Six months later returned complaining of a loaded feeling after food and giddiness on first getting out of bed. Chest now for the first time shewed some dulness over upper right lobe, but not quite so

Case 12



Case 12.



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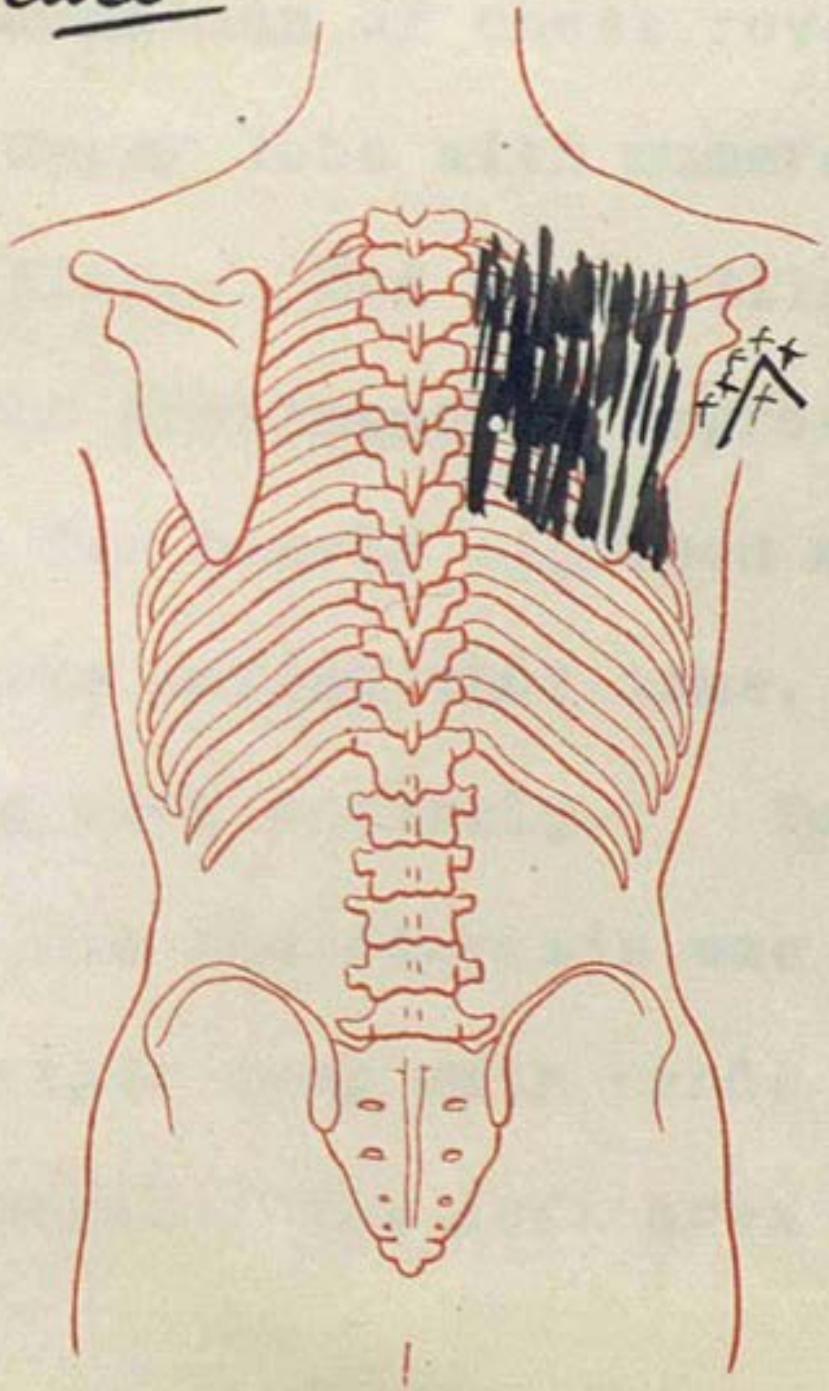

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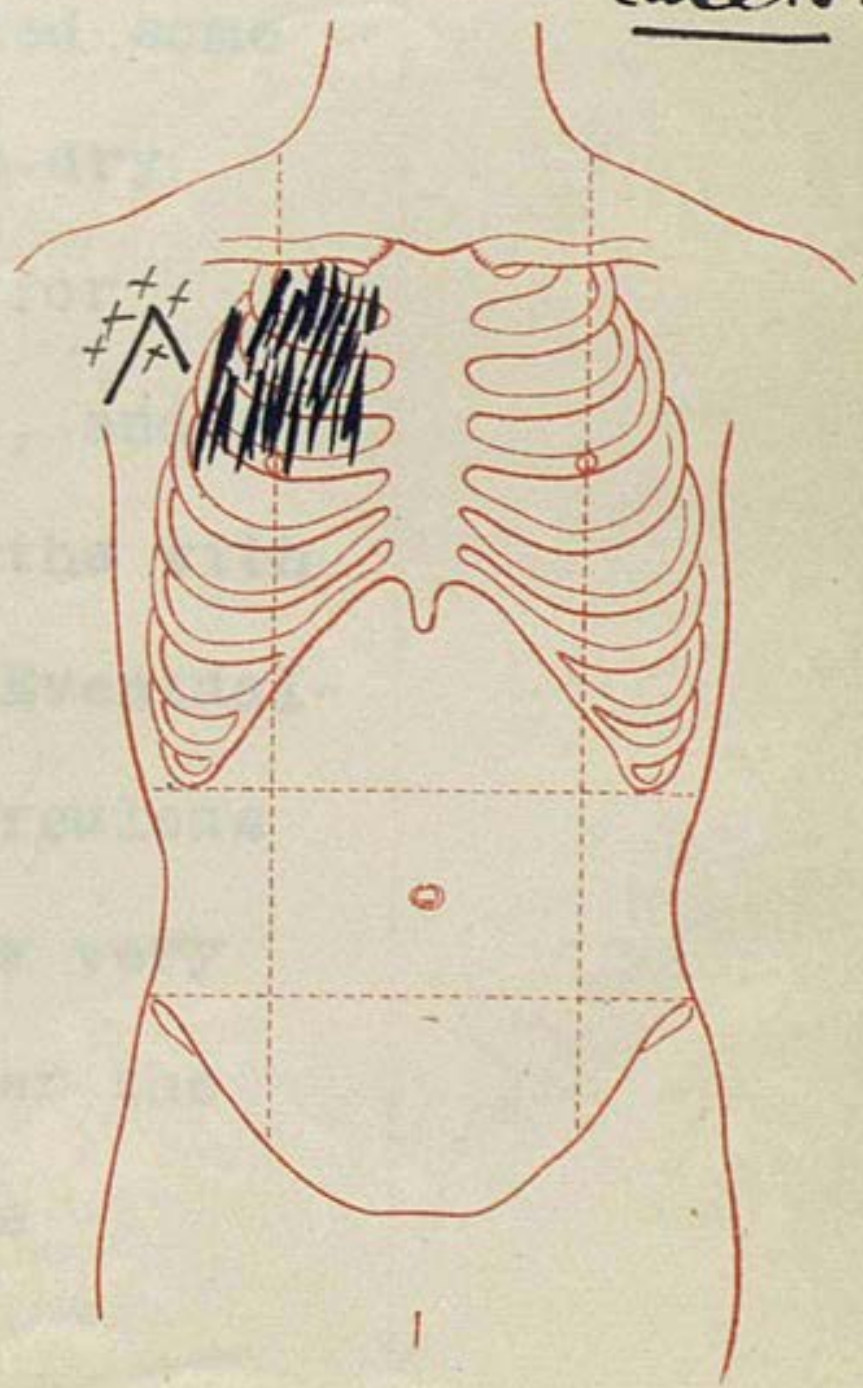
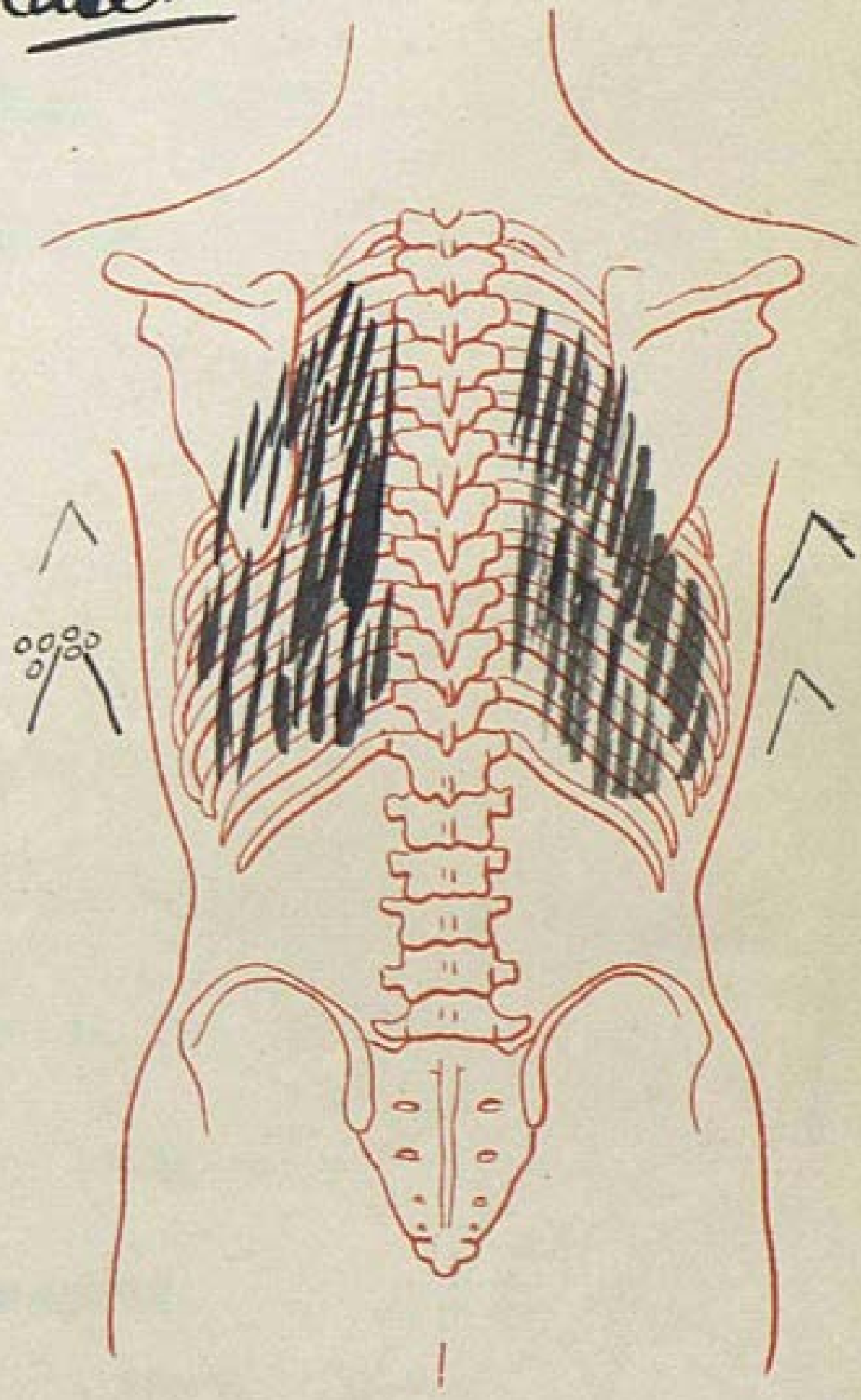


absolute as the shading in diagram would imply. He had now slight cough and was losing weight. He was sent to the country, and during the course of six weeks patient gained a little over a stone in weight. He was now anxious to resume work, but was warned against doing so. He now fell into drinking habits again, but did not consult me for more than eighteen months. He was then very dyspnoeic and had a harassing cough and copious expectoration. Tubercle bacilli were found in the sputum, but were by no means abundant. As the patient thought I was not treating him properly he called in another medical man, but is now I understand slightly improved although not likely to resume work again.

Case 13. J.M., aet. 39, married. Home indifferent. Complained of dyspeptic symptoms and slight cough. Examination of chest revealed some dulness over right upper lobe with numerous dry sounds on auscultation. Had been ailing for about a year. Under treatment he improved, and was able to work on for nearly eighteen months with short periods of rests during that time. Eventually he had to give up work entirely. Tuberculous changes then set in, and the fibrosis was now very extensive over the right lung with bands over the left lower lobe as well. The left apex was

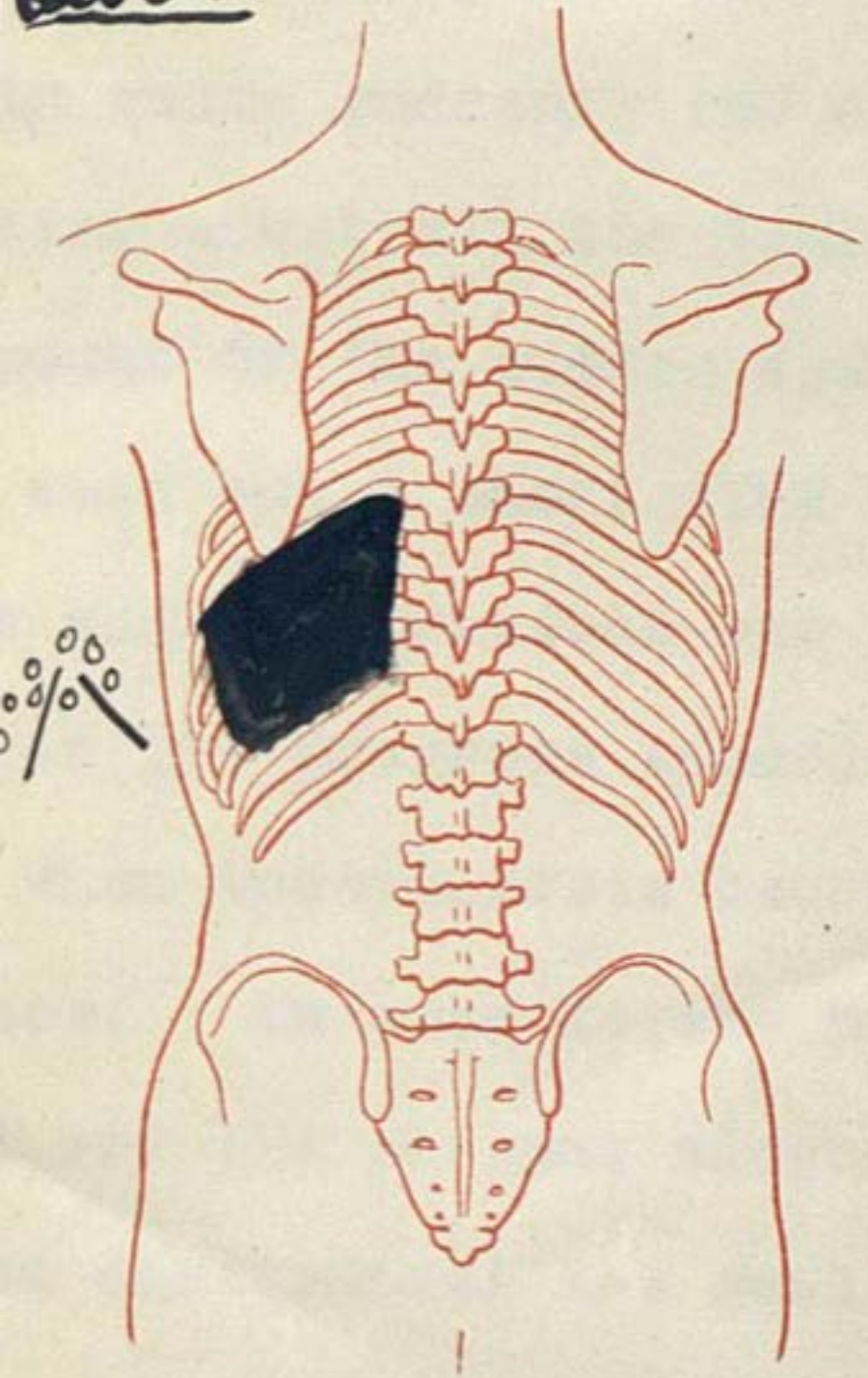
Case 14.



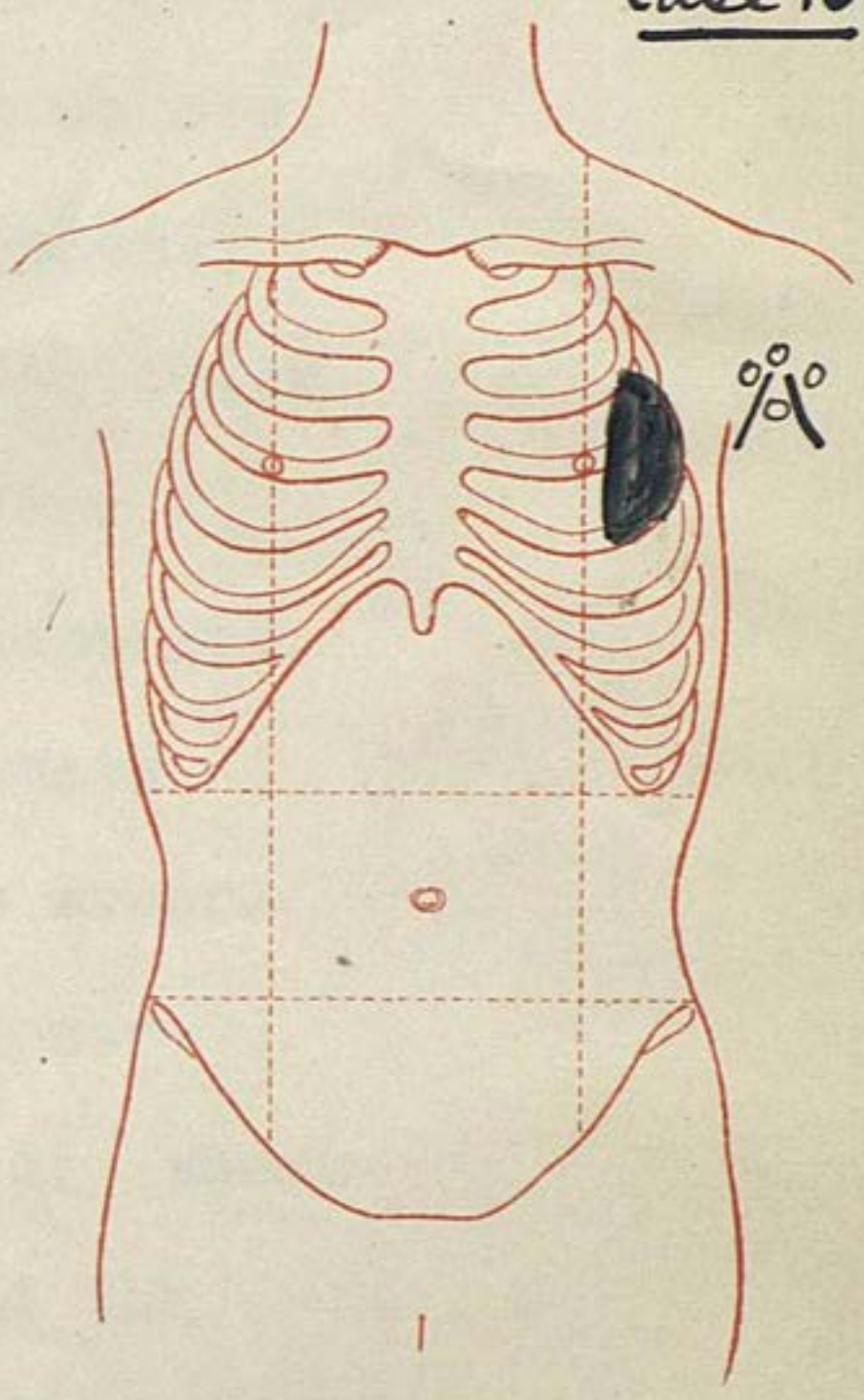
evidently breaking down. The sputum was loaded with tubercle bacilli, the urine was also albuminous, while the fingers were clubbed, the cheeks flushed and the lips cyanosed. This patient went down-hill rapidly, and died at the age of 42 from toxaemia associated with virulent diarrhoea and a troublesome bedsore over the sacrum. Well marked leucocytosis was found in the blood a fortnight before death.

Case 14. W.B., aet. 37, maried. Miserable surroundings. Lived in a dark room on the ground flat which looked out into a back yard. Alcoholic. Food bad, patient getting a good deal of tinned meats. Complaint, cough which had lasted three months. Both lungs more or less dull posteriorly. Bronchial breathing with crepitations over left base. This patient improved and went home to the North of England, but got worse there. He now took to bed altogether, and rapidly developed signs of an extensive bronchiectasis at left base. No tubercle bacilli in sputum. One day he coughed up a considerable quantity of blood. The expectoration thereafter was always more or less blood-stained. No albuminuria and no leucocytosis present. He died about eight months after I first saw him. Just before death the face became markedly flushed especially over the cheeks, and the ankles began to

Case 15



Case 15.



be a little puffy. He passed away quietly, and shewed no evidence of marked dyspnoea.

Case. 15. aet. 40, married. Home comfortable. Patient temperate. Food satisfactory. Took ill about ten months before I first saw him. Complaint, marked weakness and slight cough. Chest as in diagram. No tubercle bacilli in sputum. This patient rapidly developed tuberculosis of the left upper lobe, and died at the age of 41 from pneumothorax which set in quite suddenly two days before death. He was going about all the time, andonly took to bed on account of the sudden dyspnoea which came on as I have said but a short time before death. This case and the previous one illustrate well the rapidity of the progress towards death which takes place when tuberculosis becomes superadded in these cases. In case 14 the home surroundings were bad and the patient alcoholic; whereas in this case the environment was good and the patient temperate. These facts go to prove that even the surroundings and the habits of the patient count for little in some of these cases.

I need not refer to other cases which have come under my notice, as the brief notes of these fifteen will serve to indicate what is the general nature and progress of cases of pulmonary fibrosis as it occurs in stone-masons. I have not felt it

necessary either to give the results of my examinations of the urine and blood which were made in a number of these cases, as the table given under case 9 may be taken as fairly typical of the results which I obtained in my other cases. It would, perhaps, have been interesting to have dwelt at some length upon the relative progressiveness of the disease as shewn by the cases which have come under my observation, but to go into a full consideration of all the factors involved would, I feel, unduly prolong this monograph. Probably more will be gained by considering the principles on which the diagnosis and treatment of these cases are based than by prolonging the consideration of the actual cases themselves.

Diagnosis of Pulmonary Fibrosis.

I intend under this heading not merely to limit myself to a consideration of the diagnosis of this condition as it occurs in stone-masons, but rather to look at it from a broader standpoint. I shall, therefore, discuss the diagnosis of pulmonary fibrosis in general as well as that of stone-masons in particular.

A reference may first be made to the diagnosis of pulmonary fibrosis occurring in children. There is usually a history of pre-existing measles, pertussis, or of pneumonia. On inspection the chest

will be observed to be more or less indrawn over the area of lung tissue involved. The vocal fremitus will be diminished or even absent as a rule, while the amount of expansion will be greatly lessened on the affected side. The breathing will tend to be bronchial in type, but is often somewhat indistinct. As a rule we find accompaniments indicative of bronchiectatic dilatation. The opposite lung may shew a certain amount of emphysema, while the heart will be enlarged, and the veins on the chest wall markedly dilated. Dyspnoea will be apparent, while a paroxysmal cough with fairly copious expectoration, resembling in many respects that which is got in the bronchiectasis of adults, is usually complained of.

It is not always an easy matter to recognise the true nature of the disease in such cases, but by carefully observing that the physical signs are usually confined to one side of the chest the presence of fibrosis rather than of tuberculous disease may be suspected. Moreover tuberculous children lose flesh very rapidly, whereas this is not so when they are affected with pulmonary fibrosis, although they may be thin and spare. Displacement of organs, especially the heart which is drawn over to the affected side, is also a helpful diagnostic point. In a few cases we find no retraction of the chest

in the pulmonary fibrosis of childhood, and this should be borne in mind in connection with the diagnosis.

To sum up, therefore, the points that must be specially attended to in such cases we may say that these are as follows:- (1) Limitation of the physical signs to one side of the chest. (2) Character of the cough and expectoration. (3) Nature of the pre-existent disease. (4) Chronicity of the patient's symptoms. (5) Retraction of one side of the chest and displacement of organs towards that side.

Turning our attention to the diagnosis of pulmonary fibrosis in adults we find that this is by no means easy. Still there are certain facts which when taken together make up a fairly definite group of distinguishing features of this disease. When doubt exists at first in the mind of the physician as to the real nature of the case time usually serves to indicate to him whether pulmonary fibrosis is present or not.

At the very outset let me state that no one, however expert a clinician he may be, should ever hazard a diagnosis of pulmonary fibrosis unless he has examined the entire chest and weighed well the history and symptomatology of the case. The patient should, therefore, always be stripped for

examination, otherwise errors in diagnosis are certain to arise. Moreover inspection and palpation should not be neglected, as they too often are, in favour of percussion and auscultation which are not always absolutely reliable in such cases.

I shall now mention seriatim the various points to which special attention should be paid in forming a definite opinion as to the presence or absence of pulmonary fibrosis in any particular case that may come before us.

The disease is usually unilateral, or if bilateral both sides are practically fibrosed to the same extent. In nearly three-fourths of my cases this was found to be the case. The number in which the right lung was alone affected was 17, while there were 18 cases of purely left lung involvement. Thus it would appear that neither lung shews any specially marked tendency to be selected. On the whole, however, I think the right lower lobe will be found the part most frequently attacked by this disease. It must be remembered that my cases were all of one class and that I am now speaking of pulmonary fibrosis from whatever cause arising. When we find both apices involved at one and the same time and to exactly the same exyent we may be almost certain that the lesion is one of fibrosis rather than of tuberculous disease.

I have referred to the frequency with which the lower lobes are affected. If in such cases we find involvement of the upper lobes as well we should rather suspect tuberculosis.

Retraction of the chest is helpful, but this only occurs in long standing cases, and is not therefore a very useful diagnostic aid in cases just beginning to shew evidence of pulmonary lesion. Displacement of the heart and other organs is also of some service in diagnosis. In pulmonary fibrosis it must be remembered that this displacement when it is met with is found to be towards the fibrosed lung. On the affected side we may frequently observe that the movement of the diaphragm is more or less restricted. I have observed the same phenomenon in chronic pulmonary tuberculosis and also in some other affections, but this limitation of diaphragmatic movement is a point which should not be lost sight of in considering the diagnosis of the disease under consideration.

Again the chronicity of the symptoms is very important and helpful. Such cases often go on for years without any marked change in their symptoms, while there is often very little wasting, although this gradually, if somewhat tardily, makes its appearance.

Another fact of interest, and one which is very suggestive in these cases, is that when we come to compare the extent of the involvement of the pulmonary tissue and the symptoms presented by the patient we find that there is a distinct disproportion between these.

The absence of haemoptysis, night sweats, rapid pulse, and temperature serve to distinguish this disease from incipient pulmonary tuberculosis with which it may readily be confused. In both diseases we may find no tubercle bacilli in the sputum so that too much reliance should not be placed on this fact in diagnosis, Of course if organisms are found then simple pulmonary fibrosis cannot be stated as the diagnosis. In doubtful cases the larynx may be examined and even the X-rays may be called into requisition, while the tuberculin test may be applied; but these methods of diagnosis will be more fully set forth when I come to speak of the recognition of secondary tuberculous invasion occurring in cases of pulmonary fibrosis.

Looking more particularly at the physical signs we find that in pulmonary fibrosis the dulness tends to run in bands which are met with more especially towards the base of the affected lung. The vocal fremitus is, in my experience, usually

diminished over the dull area, but in some few instances a slight increase may be observed. The breathing as a rule is bronchial, although in some cases it may be vesicular with slight prolongation of expiration. In almost every instance, however, the breathing of whatever type it be shews marked feebleness, while we not uncommonly find the respiratory murmur absent altogether. The vocal resonance is greatly diminished and at times entirely absent over the fibrosed areas of lung tissue. This combination of physical signs at once suggests pulmonary fibrosis, although this diagnosis may not always ultimately prove to be correct. The presence of rhonchi or of crepitations always adds to the difficulty of diagnosis, and in many cases we find these accompaniments present as well. I wish to call attention once more to the curious and characteristic creaking sound which is often heard over the affected areas in pulmonary fibrosis. I cannot find a word which exactly expresses the nature of this sound, but it is of a grating character, and is not at all like coarse friction, so that to an experienced and careful auscultator there is no fear of its being mistaken for the latter. I have detected it in a large number of cases, and I now rely on it as a guide to diagnosis in otherwise uncertain cases.

In pulmonary fibrosis, when the disease has lasted for some time we find more or less emphysema present in the opposite lung as well as in the unaffected parts of the fibrosed organ. Of course we may also have the signs of chronic bronchitis and of bronchiectasis present as well, but these need not be further referred to, although their presence always renders the diagnosis more difficult and uncertain.

Passing to the diagnosis of pulmonary fibrosis as it is met with in stone-masons we have less difficulty as the knowledge of the patient's occupation affords a clue to the real nature of the disease. I wish, however, to state most emphatically that pulmonary fibrosis may actually exist while as yet there are little or no pulmonary symptoms present. In fact, as I have already pointed out in considering the symptomatology of this disease as it affects stone-masons, dyspeptic symptoms are often the first to be complained of by the patient himself, while weakness, vertigo, or faintness may exist for some time before the presence of cough dyspnoea or expectoration is observed by the patient. When a stone-hewer comes to me complaining of dyspeptic troubles and general debility I make a point of examining the entire chest, and even if I detect no physical signs of disease at

the time I note the case and expect that sooner or later the pulmonary fibrosis will give more distinct and characteristic evidence of its presence. There is usually no doubt as to the real nature of the disease when a stone-hewer comes to us complaining of shortness of breath and by and bye has symptoms of a cold or complains of a cough. With the latter there is, however, at first no expectoration. It is a characteristically dry cough, and on auscultation of the chest we may be surprised to find no rhonchi or râles present.

The recognition of the engrafting of a tuberculous process in cases of pulmonary fibrosis is not always a simple matter. Of course when we find definite evidence of tuberculous disease present such a rapid wasting, hectic temperature, and sweating associated with the presence of tubercle bacilli in the sputum and it may be haemoptysis, then the recognition becomes a simple enough matter. Apart from this, however, we have to rely on certain other evidence. In not a few instances the tuberculous invasion gives rise to little or no alteration in the patient's symptoms or appearance for some time, while no tubercle bacilli may be found in the sputum even when the latter has been centrifuged and examined repeatedly. How then can we tell that a tuberculous invasion has taken place? We may not

be able to do so with absolute certainty in the absence of typical accompanying symptoms and signs; but I think a strong suspicion of its occurrence should be felt when the urine becomes albuminous, when the blood shews a leucocytosis, and when the patient's weight suddenly diminishes to a marked extent, a slight but steady diminution being much less significant. The occurrence of haemoptysis is of little value when taken alone in the recognition of tuberculous invasion as blood-spitting may result from bronchiectasis which is almost always present in the later stages of pulmonary fibrosis.

When albumen is absent from the urine and the blood count not at all suggestive then we must have recourse to other means for determining the presence of secondary tuberculosis in the absence of bacilli from the sputum, and other incontrovertible evidence. In such cases the X-rays may sometimes prove of service as may also the use of the laryngoscope; while the tuberculin test may be applied. I shall now briefly refer to each of these three special modes of diagnosis in turn.

(1) Rontgen-ray examination. This is most valuable as an aid to the early recognition of secondary tuberculous lesions. The part which is suspected to be the seat of these changes shows a distinct loss of translucency on the screen, and a marked shadow on the radiograph if taken. As a

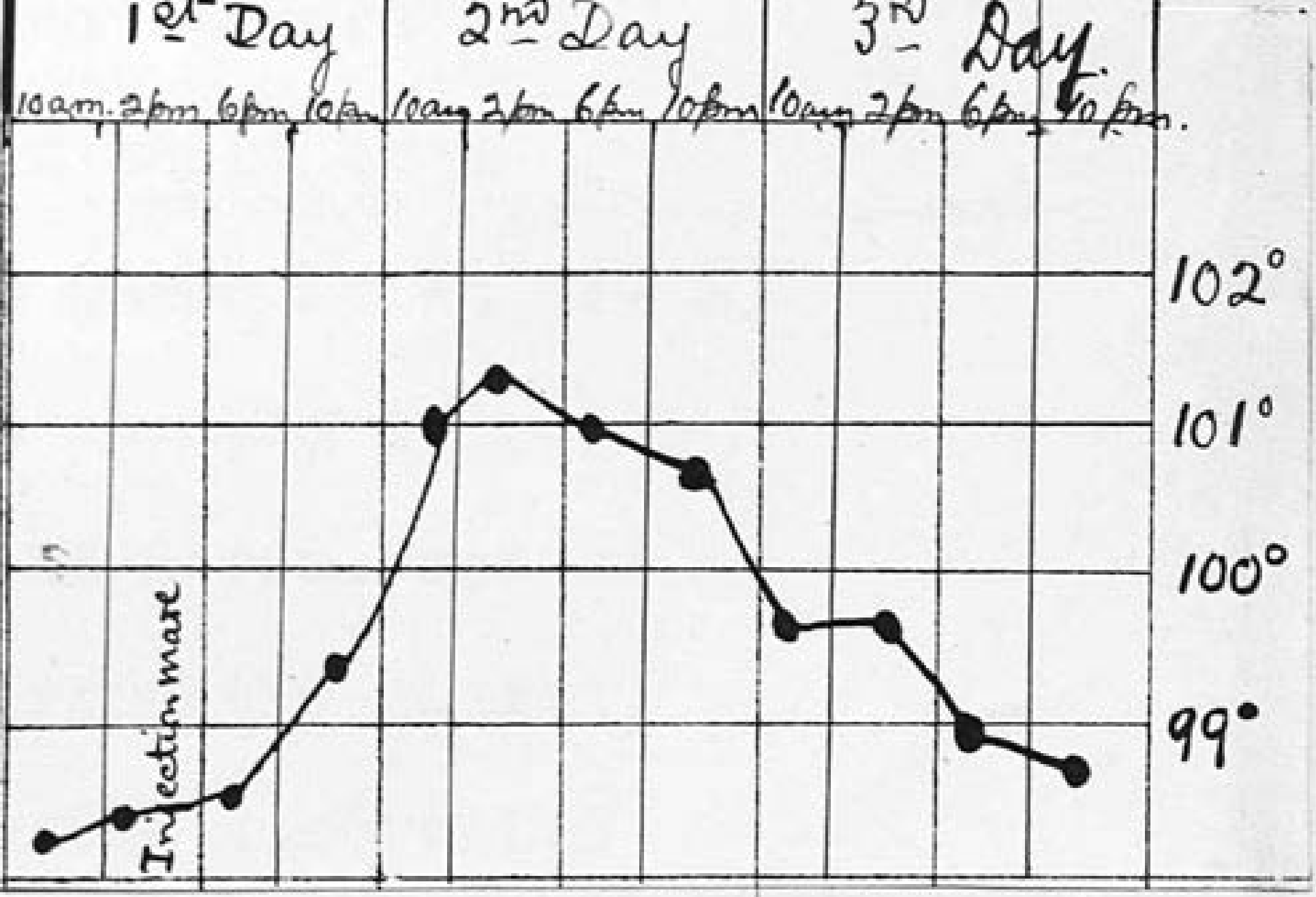
general rule the use of the screen alone gives the best results, but there is no reason why the radiograph should not prove equally valuable provided the length of exposure can be sufficiently shortened so as to prevent respiratory and cardiac movements from interfering with the picture obtained. Interference with diaphragmatic movement on the affected side is not of much importance as this may quite well occur in cases of pure fibrosis. The opacity got on the screen examination and appearing in the radiograph need not, of course, imply a tuberculous lesion, but it is suspicious when we find the patient getting suddenly worse without being able to detect any new physical signs in the chest. There is no doubt whatever that translucency will be got on the screen in many cases long before the affected part gives evidence of disease on physical examination.

One thing is necessary in carrying out an X-ray examination in these cases. It must not be undertaken hurriedly. Considerable experience is also necessary for the interpretation of the results of such an examination, but in the hands of a trained observer this method is of undoubted value in the early recognition of tuberculous changes occurring in cases of pulmonary fibrosis.

(2) Laryngoscopic examination. This is most important, and it is advisable that a careful

examination should be made in all suspicious cases. The palate and epiglottis may be pale, as may be also the larynx in general, while there is usually a want of tone in the parts. This anaemic condition of the larynx is very suggestive, and if found present it renders the diagnosis of tuberculous changes in the lung almost certain. As a rule, however, the larynx is normal even when the lungs are involved in a tuberculous process. Nevertheless we should not omit to examine the larynx on this account, as even a negative result may be of some little value in such cases.

(3) The Tuberculin test. In carrying out this test a one per cent solution is employed. The injection may be made into the muscles of the back, and the amount used should be .001 c.c. Certain precautions must be taken before deciding on the employment of this test, which I regard as perfectly safe if due attention is paid to the following points:- The patient's temperature should be normal and he should not be acutely ill at the time of injection. The presence of haemoptysis even in slight amount I always regard as a contra-indication to the employment of this test, as also severe bronchitis. If we obtain a rise of temperature after the injection we may safely diagnose the presence of tuberculous invasion. If there is no


1st Day
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102°
101°
100°
99°
Injection made

reaction we may reasonably wait for a day or two, and give a second injection, this time doubling the dose. When a positive reaction is obtained the diagnosis then becomes certain, and although in a small minority of instances a reaction may be obtained even when no tuberculous lesion is present, the fact holds true for most cases that a positive reaction may be relied on with almost absolute confidence. I have employed this test in one or two cases of pulmonary fibrosis in which I suspected a secondary tuberculous invasion, and in every instance obtained a definite reaction after the first injection; while none of the patients were adversely influenced by the use of this test. In every case the results obtained were borne out by other evidence which, however, only made its appearance at a considerably later period. After the injection I usually noted the patient's pulse and temperature every four hours during the day. A chart of a case in which I used this method is given as an illustration of the reaction which is usually to be obtained. The injection was made at 2 p.m. and at 10 p.m. the temperature was very distinctly raised, and exactly twenty-four hours after the tuberculin test was applied the thermometer registered the highest, viz. 101.4° Fahr. Thereafter the temperature fell until the normal level was again

reached on the morning of the fourth day.

Cases will frequently occur in practice in which the physician may be in doubt as to the Differential Diagnosis of the diseased condition present, and may be unable to determine in what way the physical signs obtained are to be ~~deter~~ interpret-ed. ~~mined~~. Pulmonary fibrosis may simulate quite a number of morbid conditions. I shall now briefly refer to these, and point out in what respects they chiefly differ from pulmonary fibrosis as regards their physical signs. The following conditions may all more or less resemble pulmonary fibrosis on physical examination:-

(1) Chronic fibro-tuberculosis, that is pulmonary tuberculosis in which we have chronic fibrosis occurring. Fibroid phthisis is the name usually given to this condition, but one to which there are very serious objections. The differential diagnosis here is often beset with difficulties. Emphysema and bronchitis may be present, but the facts that the apices are more frequently attacked than the bases and that repeated examination of the centrifuged sputum usually reveals a few tubercle bacilli are helpful. The history of the patient is also important, as in the case of fibro-tuberculosis we find at the beginning night sweats, swinging temperature and it may be haemoptysis. At times

also these symptoms recur at short intervals. Otherwise there is little that distinguishes a pulmonary tuberculosis in which fibrous change has occurred from a case of pure pulmonary fibrosis.

(2) Pleurisy with Effusion. Here we may be unable to diagnose between this condition and that of pulmonary fibrosis. In cases of pleuritic effusion the organs are pushed over to the opposite side; whereas in fibrosis they are drawn over to the affected side by the contracting lung tissue. As a rule, too, we find a much shorter history in the one case than in the other; moreover the dulness in pleuritic effusion is more intense and absolute than in pulmonary fibrosis. Above the level of the fluid we find aegophony, which is not obtainable in fibrosis. Again in cases of pleurisy we are not so likely to get rhonchi and râles, nor yet the characteristic creaking sounds which are so frequently present in fibrosis of the lung. As a rule, when still in doubt, it is advisable to put in an exploring needle so as to clear up the difficulty.

(3) Thickening of the Pleura. The only way in which I think we can distinguish between a simple pleural thickening and pulmonary fibrosis is by obtaining characteristic creaking sounds over the dull area in the latter condition. We are also far more likely to have emphysema and bronchiectasis in

cases of fibrosis. Early cases may be a source of difficulty, but when fibrosis is in its initial stage I do not think the dulness is nearly so great, nor the interference with vocal resonance and fremitus quite so marked as in pleural thickening.

(4) Collapse of the Lung. Local areas of pulmonary collapse may simulate fibrosis, but in the former condition there are no accompaniments such as rhonchi and râles present, nor is there any displacement of the heart and other organs. Sometimes the history of the case is also a help to the diagnosis in these cases.

(5) Bronchiectasis. This may of course complicate pulmonary fibrosis. When, however, it exists apart from the latter condition we may have some difficulty in differentiating the two diseases. We rely on two points mainly for the diagnosis in such cases, viz., the amount of dulness and the history of the case. There is practically little dulness to be obtained in cases of simple bronchiectasis, while the history is one of fairly acute onset with a definite illness to account for the condition.

(6) Malignant disease of the Lung or Mediastinum In this condition we find cough, dyspnoea, with it may be wasting and haemoptysis. There is also a greater or less amount of expectoration. For the

differential diagnosis we must rely chiefly on the presence or absence of pressure phenomena. These are common in malignant disease, whereas they are non-existent in cases of pulmonary fibrosis. When, however, pressure effects are not yet present we must rely on the nature of the physical signs, but even these are not always of much value. In cases of tumour the dulness will usually be more striking and more extensive, while it never occurs in bands. It will not tend to be so much localised to the bases, while rhonchi and rales in association with a bronchial type of breathing would be more suggestive of fibrosis than of tumour. Otherwise I do not know of any point which can really be taken as a reliable guide to the differential diagnosis of these two conditions. Even after the greatest care in examining the chest a wrong diagnosis may be arrived at, and at the autopsy a tumour of the lung may be found occupying an area which was supposed to be simply the seat of fibrosis. If the disease has lasted for some time it is more likely to be fibrosis, especially if the symptoms have not progressed, and the patient's weight has remained practically stationary during that period. The presence of emphysema or of bronchiectasis, moreover, would be in favour of pulmonary fibrosis.

(7) Aneurism, (especially of the thoracic part

of the aorta). Here we rely on the presence of pressure symptoms for the differential diagnosis, as well as on the history of the case. Usually a patient suffering from aneurism is a powerful man with no retraction of any part of his chest and no emphysematous change, while dull areas at the bases are not likely to be met with. It is in the early stage that we find most difficulty, for even an aneurism may at first give rise to little trouble unless it be slight cough or some dyspnoea on exertion. Careful examination of the praecordial and aortic regions will usually, however, reveal some abnormality suggestive of aneurism rather than of pulmonary fibrosis.

(8) Syphilitic Pulmonary Fibrosis. The physical signs may be the same in this as in simple fibrosis, and we can only differentiate the two by obtaining a history of definite infection and by the presence of other syphilitic manifestations. When we cannot obtain a definite history and when no other evidence of syphilis is present it becomes, of course, absolutely impossible to distinguish this from the simple or non-syphilitic form of pulmonary fibrosis.

(9) Pulmonary Tuberculosis (especially the basal form). The differential diagnosis of the two conditions is sometimes impossible for a time. If the condition is found at the apex, the diagnosis is in

favour of tuberculosis. It is the basal cases that present most difficulty. We rely upon the presence of hectic temperature, night-sweats, rapid loss of flesh and non-existence of anything like the marked emphysema got in pulmonary fibrosis, and above all upon the presence of tubercle bacilli in the sputum. If both apices are involved to the same extent, and that but slightly, we may almost certainly negative the possibility of the lesion being a tuberculous one, while a basal tuberculous condition is generally found to be associated with a certain amount of apical disease.

From what I have said regarding the differential diagnosis of fibrous hyperplasia of the lung it will be gathered that it is not always an easy matter to decide as to the real nature of the case. When met with in stone-masons, however, as already pointed out, there is comparatively little difficulty in arriving at a correct diagnosis if we keep in view the nature of the patient's occupation and the symptoms which specially characterise this form of pulmonary fibrosis. It is when complications set in in such cases that difficulties arise, for then we may be unable to tell how far the symptoms and signs are the result of the original fibrosis and to what extent they are produced by the secondary complications which arise in consequence of this disease.

The Treatment of Pulmonary Fibrosis Occurring in Stone-Masons.

In considering the treatment of this disease it is of the utmost importance that we should refer very carefully to its Prophylaxis. The old proverb "prevention is better than cure" is one which may truly be quoted in connection with pulmonary fibrosis as it is met with in stone-hewers. Once the disease has set in it is by no means easy to check its progress, much less to eradicate it. It may be asked, Can we prevent a disease which is undoubtedly associated most intimately with the patient's work? I believe we can, provided we obtain the necessary co-operation on the part of the workman and his employer.

In the first place if the pulmonary fibrosis of stone-masons is to be abolished we must get rid entirely of closed-in sheds. These are veritable death-traps. There is no reason why stone-hewing should not be carried on under cover, but the roof should rest on iron pillars, and not on walls of wood or of brick which close in the working space completely. It would be well too if some means could be devised whereby the dust would become wetted by some material thrown on it in the shape of a spray. The dust would then tend to fall to the ground and cease to be inhaled by the workers. The

difficulty in carrying out this treatment of the dust is that it is well nigh impossible to moisten the dust without at the same time wetting the workmen.

A much more readily adopted plan whereby dust inhalation may be prevented is to compel the workmen to wear respirators. These may be constructed in various ways, but they must be light and at the same time impervious to dust; while they should be large enough to cover over the orifices both of the mouth and of the nose. Workmen, unfortunately, absolutely refuse to wear such appliances, and even compulsion would in some cases lead to strikes, or at all events to friction between the workmen and their employers without the latter gaining anything in return for their trouble.

Failing a properly constructed shed, special treatment of the dust laden atmosphere, and the use of suitable respirators we must see that the general conditions of the workman as regards his food, drink, and habitation are satisfactory.

I have already referred to the diet of stonemasons, and to their custom of carrying most of the food to their work. This "carried food" is for the most part indigestible, and tends sooner or later to produce evidences of chronic dyspepsia. The remedy is simple, and consists in providing properly

cooked meals at reasonable prices to the workmen on the spot. Then again even the food taken at home is not always of the best, nor is the cooking of it satisfactory. Tinned foods, condensed milk, and tea enter largely into the diet list of the stone-mason. Fresh beef and vegetables are often never paratken of from one year's end to another, while foods made with milk are seldom if ever seen on his table. No doubt this improper feeding weakens the tissues and makes them an easier prey to disease.

Alcoholic indulgence is unfortunately all too common amongst stone-masons, and if we are to adopt prophylactic measures against pulmonary fibrosis we must certainly see that these workmen adopt temperate habits.

Lastly their dwellings are often dark and otherwise unsanitary. The windows are kept closed both by day and by night. Dust is allowed to accumulate everywhere, and often a large family will be found crowded together in a small house consisting of but one small room and kitchen. Even in localities where houses are built on hygienic principles this class of workmen will do all they can in their power to secure that fresh air never enters and that even the sunlight is obscured by windows coated with dust and dirt. All this must of necessity favour the inroad of disease, and prevent

its successful treatment when once it has laid its hold upon the workman.

Prophylaxis, however, is a much simpler matter to write about in theory than to carry out in actual practice. Accordingly the preventive treatment of pulmonary fibrosis need not be further referred to. I shall now therefore consider the actual treatment of the disease, mentioning more especially those measures which I have found helpful in my own practice.

At the very outset let me emphatically state that every stone-hewer should give up this work when he reaches the age of thirty-five. The "critical epoch" is between the ages of thirty-five and forty years. At this age he should change his occupation. This, too, is a somewhat theoretical recommendation, for it seldom happens that a stone-hewer can find other work to do. If he can, however, he should select work which will keep him out of doors as far as possible. Further, if he can obtain employment in the country so much the better. The new work, however, must not be too hard or over-fatiguing. A question often asked by these workmen is, "Should I emigrate?" And this is not always an easy question to answer. Emigration would be highly commendable in such cases provided we could be sure that the workman would find

suitable employment abroad. Too often he goes out to Africa,,or to Canada, only to find that there is no room for him. Accordingly we should pause before recommending such patients to leave this country, unless they have friends in the Colonies to whom they can go and on whom they can for a time at least depend.

Apart from all this, whenever the patient shews the first evidences of pulmonary fibrosis he must be put into the most favorable conditions as regards diet, rest and so on. The stomach must not be overloaded with much fluid nor yet with fermenting foods. Dyspeptic symptoms are most quickly relieved by careful dieting, the avoidance of "carried food", and by the administration of acid tonics after meals combined if necessary, with glyceole of pepsin. General debility, so often present in these cases, is often greatly benefited by rest and good feeding together with the use of strychnine and some readily assimilated form of iron, such as ovoferrin or one of the newer haemoglobin preparations.

When evidences of cardiac weakness are present a combination of strychnine with strophanthus is often found serviceable, while in conditions of serious heart failure with oedema of the ankles and other attendant phenomena these remedies may be

given hypodermically. Vertigo and syncope are often met with, and when these are present rest must be insisted on. Even when no heart symptoms are complained of this organ should be toned up by judicious dieting, periods of rest, and general tonic treatment. Attacks of syncope are frequently the result of anaemia, a fact which is of importance from a therapeutic point of view.

Bronchial catarrh is an indication for the use of such remedies as ammonium carbonate and strychnine, which strengthen the respiratory muscles and so aid in the expulsion of the somewhat tenacious and scanty sputum. Bronchiectasis necessitates the use of creosote in addition, in one or other of its various preparations such as guaiacol carbonate. Personally I prefer to give it in the form of beechwood creosote combined with maltine.

When the cough is troublesome sedative mixtures are sometimes indicated. Disinfection of the sputum is advisable in all cases, and should be insisted on by the medical attendant. For the dyspnoea I know of only two remedies which are of any value, viz., potassium iodide and strychnine. The former must be given in fairly large doses, best combined with ammonium carbonate. Haemoptysis, when copious, necessitates the use of morphia and the patient must be kept absolutely at rest.

Diarrhoea in the later stages may be controlled by ichthoform (a most excellent remedy in such cases) or by the use of morphia suppositories.

Insomnia may be troublesome, but hypnotics must not be given in the first instance. We must endeavour to obtain sleep for the patient by the use of hot baths, proper ventilation of the sleeping apartment and other simple measures.

Thus far I have mentioned briefly certain indications for treatment in this disease; but it is necessary to go further and to lay down certain definite principles which should guide us in our management of cases of pulmonary fibrosis occurring in stone-masons. As my experience of this malady has ripened I have been more and more led to adopt the following measures in every case. Of course it has not been possible to carry them out completely in all the cases that have come under my notice, but one should aim at their adoption as far as possible.

(1) The general nutrition of the patient must be maintained at as high a level as possible; and loss of weight must be combated by every available means in our power.

(2) The heart must be strengthened to withstand the effects of backward pressure and engorgement which are apt to embarrass it when the

pulmonary tissue becomes seriously involved.

(3) Bronchial secretion must be got rid of, and coughing facilitated as only thereby can we hope to prevent bronchiectatic dilatation from supervening. At the same time any bronchial catarrh that may from time to time be present should be treated very actively with a view to lessening the amount of emphysema as well.

(4) The respiratory muscles must be strengthened in order to make up for the loss in the respiratory capacity of the lungs.

(5) Invasion of the lungs by the tubercle bacillus must be strenously guarded against.

In order to maintain the general nutrition of the patient various measures may be employed. First and foremost must be placed good feeding. The diet should consist largely of fresh eggs (preferably raw), milk, cream, butter, soups, and a sufficiency of butchers' meat together with fish and, wherever available, fowl. In addition cod liver oil and virol will be found of undoubted value. There is one drug which in my experience seems to aid nutrition and also to affect metabolism to such an extent that a patient previously losing weight begins to gain during its administration. The drug I refer to is Ichthyol. My experience with this remedy in pulmonary affections formed the subject

of a paper read before the Therapeutical Society of London in 1903, and published in the Lancet (August 8, 1903). It may be well if at this stage I briefly refer to my experiences with ichthyol in the treatment of pulmonary fibrosis.

I am of opinion that ichthyol acts feebly as an antiseptic, but is more specially valuable on account of its action upon nutrition and also because of its local effect. During the course of my observations I found that patients who were taking ichthyol shewed an increased appetite for food and a distinct gain in weight. Ichthyol, therefore, must influence nutrition. Many authorities maintain that ichthyol owes its beneficial effect largely to the amount of sulphur which it contains. Now sulphur has but little physiological action on the bodily organism. It is chiefly an alterative, although it does to a certain extent modify nutrition. But it must be kept in mind that ichthyol is more than sulphur; it is a definite chemical compound and as such has a definite action upon the general bodily nutrition. In my own mind there exists no doubt whatever that ichthyol is a conserver of nutritive force. It seems to have the power of checking waste in the bodily economy which is no doubt due to the sum-total of all the elements composing the drug. Under its continued administrationthe nutritive force of the tissues is

generally raised; and hence among other beneficial results they are enabled to withstand more readily the action of toxins, or at least the latter have a less powerful and destructive effect upon the tissues.

Again as to the local effect of ichthyol I think there can be no doubt. In erysipelas and in articular rheumatism ichthyol when locally applied rapidly brings about a reduction of the inflammation and a return of the parts to normal. So too in gynecological conditions ichthyol pessaries rapidly remove inflammation and congestion of the pelvic organs, because this agent is, par excellence, a vaso-constrictor. In pulmonary disease also ichthyol gradually reduces any inflammation that may be present. It must, therefore, be regarded as a definitely anti-inflammatory agent when applied locally, and none the less so when administered internally in sufficiently large doses.

If we look for a moment at the practical results which I have secured from the use of ichthyol in pulmonary fibrosis I think it must be admitted that considerable benefit has been derived from its administration. It must, however, be given in fairly large doses. I always give it in capsule form, and begin with 8 grains four times a day, gradually increasing the amount until 20 grains are taken with

a like frequency, this being my maximum limit. In other words the patient gets from 32 to 80 grains a day. This may seem a somewhat large amount, but it is usually well tolerated if given in the form of capsules. It practically never causes nausea or sickness when administered in this way, and is in fact often better borne than such drugs as creosote.

Generally speaking little effect is produced by the administration of ichthyol for some weeks. In fact none of my cases improved to any appreciable extent until three months had elapsed. Then, as a rule the appetite began to improve and a distinct increase in body weight was accorded. Not only so but the cough became less harassing and the expectoration, when at all abundant, became less in amount and easier of expulsion than before. In one or two of my cases a gain of from 10 to 12 lbs. was recorded at the end of six months of treatment by means of ichthyol alone, without any change in the diet.

I was specially gratified by the improvement which the administration of ichthyol effected in those cases which were complicated by bronchiectasis. In this connection I may cite one case in particular. The patient had a paroxysmal cough with abundant expectoration of a foetid character. He was losing weight very rapidly and had a remittent temperature.

The breathing over the affected area was of a distant tubular character, the tubes being evidently full of secretion. After a fit of coughing and free expectoration the percussion note was almost tympanitic, while the breathing became more markedly tubular and was accompanied by very typical coarse metallic rales. My aim in bronchiectasis has always been to obtain a regular and free expulsion of the foetid material which blocks up the tubes and tends to produce a more or less general toxaemic condition. In the patient's case referred to I found ichthyol accomplish this in a most satisfactory manner. After three weeks' treatment the cough became much less paroxysmal, and in seven weeks time both the cough and the expectoration were much less troublesome, while the latter had practically lost its foetid character. Not only so but the patient had put on five and a half pounds in weight, while the appetite was distinctly improved and the temperature was perfectly normal. The physical signs at the same time encouraged me to believe that the bronchiectatic cavity was no longer a reservoir of foetid secretion.

I think we must not underrate the value of ichthyol in aiding nutrition in cases of pulmonary fibrosis. I am firmly convinced of its efficacy in this direction as also of its anti-inflammatory effects in this disease. While, therefore, good

food must always hold the first place there is no doubt that ichthyol, given in sufficiently large doses and for a prolonged period, will be found a very valuable aid to the maintainence of bodily nutrition.

The use of raw meat in these cases has also formed the subject of personal investigation. I find, however, that stone-masons do not readily fall in with the idea of such feeding. They are apt to regard it in the light of a ridiculous experiment. I must emphasise the fact that the results obtained from feeding lower animals on raw meat must not be taken as an indication that they will hold true for the human species as well. We must give the patient what he is able thoroughly to digest and to utilise for the purposes of his bodily metabolism. It has to be borne constantly in mind that the gastric organs of these patients are not always in a healthy state, and accordingly great care has to be exercised as to the food selected. I have been disappointed in my results obtained from raw meat feeding in cases of pulmonary fibrosis. True, for a time, gain in weight was manifested; but often with concomitant gastro-intestinal disturbance. Diarhoea was common, while considerable nausea was generally complained of, and actual vomiting resulted in one case. Excess of phosphates was often present in the urine, that is to say the amount excreted was considerably greater

after the use of raw meat than before its employment, while the total acidity of the urine was apt to become appreciably lessened. On the other hand, without any increase in the total amount of urine excreted, the urea eliminated was frequently found to be very much increased. The blood counts were not more satisfactory after raw meat feeding than after the adminsitration of an easily assimilated form of iron, such as ovoferrin or one of the haemoglobin preparations. On the whole, therefore, I am not favorably impressed with the advantages, held out by certain writers, which are supposed to be derived from this method of maintaining nutrition. In fact, in spite of statements made by brilliant climicians to the contrary, I believe raw meat feeding does more harm eventually than good and should not be prescribed in such cases.

I personally much prefer a modified form of raw meat feeding which introduces it merely as an occasional item in the daily menu. In the majority of the cases of pulmonary fibrosis which have been under my care I have tried to maintain a dietary consisting largely of eggs, milk, bread and butter and vegetables, with thin raw meat sandwiches taken with cocoa in the afternoon. Butcher's meat and fish were of course also included in the dietary, but did not take so prominent a place as the other articles

mentioned. I have been perfectly satisfied with the results obtained, and see no reason why we should substitute raw meat for the other food substances mentioned. I am convinced that too much red meat in these cases is apt to prove harmful by increasing the amount of uric acid in the tissues and by loading the circulation with products of a deleterious nature. In some of my cases marked improvement in the attendant symptoms was manifested when a diet of eggs, milk and vegetables was substituted for one of meat. The dyspnoea became less marked, while the general condition of the patient at the same time shewed a definite improvement.

When a patient begins to lose weight we should endeavour by means of judicious dieting and the administration of cod liver oil and perhaps of ichthyol to increase it. It is wonderful how much weight some of these patients can be made to put on by adopting these measures, which will also be found beneficial in ameliorating certain of the attendant symptoms such as cough. So long as the patient's weight is maintained the prognosis is favorable; but when it decreases, and more especially when attempts to restore it once it has fallen off are fruitless, then the outlook for the patient becomes much less hopeful. One word of warning is necessary, however; we must carefully avoid giving the patient more proteid than he can assimilate, as

the excess would tend merely to produce an increase in the amount of decomposition products in the intestinal tract, a result which would inevitably lead to toxaemia and consequent weakening of the organism.

The second indication for treatment in cases of pulmonary fibrosis is not less important than the one we have just been considering. The heart has to be strengthened, and this is a matter which I fear is often greatly neglected in these cases. Even in the earlier stages of the disease ~~the~~ due attention should be given to the state of the heart and circulation. By attending carefully to the feeding of the patient, and also by means of judicious and carefully regulated exercise much may be achieved. In the later stages, however, it becomes necessary to administer cardiac tonics such as strychnine, strophanthus and diffusible stimulants such as ammonium carbonate. Apart from these, however, much may be done to prevent undue strain upon the heart and circulation by guarding the patient against bronchial catarrh. Such attacks tend to weaken the heart, and eventually lead to its dilatation, apart altogether from the deleterious influence which the almost inevitable emphysematous condition of the lungs has upon the organ.

These patients must be carefully guarded against chills. This is best accomplished by sponging the chest every morning and night with cold water, and

by the avoidance of overcoddling. Chest protectors should never be countenanced, and the feet should be well protected by thick-soled boots, not shoes. The house should always be kept well aired,,and the window of the sleeping apartment should be kept widely open by night as well as by day. It frequently happens that stone-masons get wet in returning home from work, and in such cases the clothes should be immediately changed and warm soup or milk taken. It is neglect of these rules which leads to bronchial troubles, while these in turn have a most pernicious influence upon the heart and circulation.

In the next place when bronchial catarrh, with cough and expectoration, is present we must endeavour to facilitate the expulsion of the secretion, otherwise bronchiectasis is apt to supervene as well as emphysema. In ichthyol we have an admirable remedy which renders the cough less troublesome and facilitates the expectoration. Other remedies are iodides and ammonium carbonate. In my hands, however, I have found ichthyol more satisfactory than any other agent. When bronchiectasis threatens it is often a good plan to raise the foot of the bed to the extent of 10 or 12 inches. This prevents the gravitation of the secretion in the tubes and leads to its rapid expulsion. The patient does not always at first take well to his new position, but in a very short time the relief afforded is so great

that he appreciates its beneficial influence. I have seen a patient extremely dyspnoeic and constantly expectorating quantities of mucopurulent material with considerable difficulty in the upright position, who was almost immediately relieved after the foot of the bed was raised. There is then much less difficulty in getting rid of the secretion from the tubes, and in consequence the dyspnoea gradually becomes less extreme.

Fourthly, in every case of pulmonary fibrosis we have a greater or less defect in the respiratory capacity of the lungs. This must be compensated for by strengthening the respiratory muscles. This may be brought about by the administration of strychnine as well as by cold sponging and friction of the chest. It is a noteworthy fact that patients affected with pulmonary fibrosis shew considerable deficiency in respiratory expansion. I have time and again observed considerable improvement result from the following course of treatment when faithfully carried out by the patient. On first rising in the morning the patient sponges his chest well over with cold water and then spends at least five minutes in friction of the skin with a rough bath towel. Thereafter olive oil is well rubbed in. This process is repeated at bedtime. In addition five minim doses of liquor strychnine is administered in a little glycerine and water thrice daily.

By this means the respiratory muscles are undoubtedly strengthened and the general condition of the patient considerably improved.

A mistake frequently made in enjoining open air exercise for these patients is to order them to take long walks. This is generally inexpedient. The patient should be warned not to walk too fast nor yet too far lest he become fatigued and his dyspnoea thereby increased. Otherwise, when taken in moderation, walking is of considerable value in strengthening the chest muscles, especially if the patient is enjoined to walk with shoulders thrown well back and the chest kept expanded by the patient taking as full inspirations as possible as he goes along.

Lastly we have to guard against the invasion of the lungs by the tubercle bacillus. Stone-masons who have developed pulmonary fibrosis seem specially prone to tuberculous infection of the pulmonary tissue. To obviate this any bronchial catarrh that may exist should be carefully and promptly treated, and bronchietatic cavities kept as free from stagnating material as possible. Otherwise we must rely on good food, abundance of fresh air both by day and by night, and general hygienic measures. I have frequently observed tuberculous changes become superadded in cases where the patient neglected recurring attacks of bronchial catarrh,

was in the habit of drinking heavily, and sleeping in a room which was never properly ventilated and in which the window was kept closed during the night. I have also known pulmonary tuberculosis set in after influenza in patients suffering from fibrosis, and in some instances an attack of acute pleurisy seems to form the starting point for the tuberculous process. The latter constitutes a grave complication, and the patient should be warned of its possible occurrence if he does not take extreme measures to prevent it.

If these general principles are attended to in the treatment of pulmonary fibrosis I feel sure that much good will result. The difficulty always lies in getting the patient to carry out the treatment. Too often these patients do not believe their lungs are affected at all, and therefore cannot be induced to adopt measures for the relief of a condition which they are convinced does not really exist. I know of no more intractable class of men than stone-hewers. They absolutely refuse to protect themselves, although they are every day face to face with living examples of the disease in those of their fellow-workmen who are still able, it may be with difficulty, to carry on their employment.

SUMMARY.

The term pulmonary fibrosis should be confined to cases which arise apart altogether from the action of the tubercle bacillus. This disease may be due to a variety of causes, but the one specially considered is that form which occurs in stone-hewers. It is due to the inhalation of dust particles, and its onset is often aided by improper diet, alcoholic habits, and unhygienic home surroundings. As a rule the patient has been previously healthy, while the family history is uaually free from any tendency to pulmonary disease. The critical epoch in the life of a stone-hewer is between the ages of 35 and 40, and no workman should continue at this trade after the age of 35.

Pulmonary symptoms are not always observed at the onset of the disease. Dyspepsia and general debility are often more prominent than cough and dyspnoea. There are three stages, that of onset in which the patient can continue at work with comparative ease, that of progress in which he works under difficulty, and that of complete breakdown in which he is compelled to leave off work entirely. The patient rarely lives beyond the age of 40, and probably most deaths occur between the ages of 39 and 42 years. Death is often the result of secondary tuberculous infection, of heart failure,

or of some other complication such as bronchiectasis. Death may either occur suddenly or the patient may be confined to bed for some days or weeks before the end.

The prognosis depends to a large extent on the ability of the patient to leave off work before the disease has become too far advanced. In any particular case the prognosis will be influenced by the extent of the fibrosis, by the nature and severity of the complications, by the intensity of the dyspnoea, and by the general environment of the patient.

The diagnosis depends partly on physical signs and partly on symptoms which are more or less definite and characteristic. In every case the urine and sputum should be carefully examined and a blood count made. The invasion of the lung by tuberculous disease should be recognised as early as possible and this may be done by examination of the sputum and larynx, as well as by the Rontgen rays and by the use of tuberculin. The differential diagnosis of pulmonary fibrosis as it occurs in stone-masons is not usually difficult, but certain other conditions may resemble it very closely, and accordingly care must be exercised in weighing the results of the physical examination of the patient and in interpreting the general symptoms in any particular case.

Treatment may be prophylactic; otherwise measures must be adopted which will relieve the symptoms and tend to prolong the life of the patient. General hygiene, good food, fresh air, moderate exercise, judicious rest, and certain drugs, especially strychnine, ichthyol and iodides, are all calculated to meet the exigencies of the case. Complications must be treated as they arise, and attention ought specially to be given to the heart which should be carefully examined from time to time.

BIBLIOGRAPHY.

Addison	Guy's Hosp. Rep. 1843; and Collected Works, New Syd. Soc.
Allbrecht	Handbuch der praktischen Gewerbe-hygiene Part I. 1894.
Andral	Clinique médicale.
Arlidge	"Pneumoconiosis," Allbutt's Syst. of Med.1898. Vol.V. pp. 242 et seq.
"	Diseases of Occupations, London 1892.
"	"Diseases caused by the Inhalation of Dust." Brit. and Foreign Med. Chirurg. Rev. 1872.
Auld	Fibroid Pneumonia. London 1891.
Bastian	"Cirrhosis of the Lungs." Reynold's Syst. of Med. 1871. Vol.III. p.815
"	Path. Soc. Trans. Vol. XIX.
Bennett	Clinical Lectures. 5th Edition 1868.
Biermer	Zur Theorie und Anatomie der Bron-chien." Erweiterung. Virchow's Archiv, Bd. XIX. 1860.
Brouardel	"Pneumoconioses." Traite de Medicine. Vol. VII. 1900.
Carre	"Bronchiectasis in Young Children." Practitioner. 1891. Vol. XLVI. (I). pp. 87 et seq.
Charcot	Pneumonie Chronique. Paris, 1860.
Clark	Fibroid Diseases of the Lung. London, 1894.
Coats	Manual of Pathology. 6th Edition, 1904.
Corrigan	Dublin Med. Jnl. Vol. XIII. 1838. p. 270
"	Dublin Hosp. Gazette 1857.

Councilmann	John Hopkin's Hosp. Bulletin. Vol.II. 1891. No. 11.
Du Castel	Pulmonary Cirrhosis. 1884.
Dolèris	Archives de Therapeutique. April 1904
Ewart	Clinical Journal, 1893.
"	"Bronchiectasis." Allbutt's Syst. of Med. Vol. V. pp. 53 et seq.
Fagge and Pye Smith	Principles and Practice of Medicine. 3rd Edition 1898.
Fagge	Path. Soc. Trans. Vol. XX. 1868.
Foster and Haldane	The Investigation of Mine Air. London, 1905.
Fox	Diseases of the Lungs. p. 412. London, 1891.
"	"Chronic Pneumonia." Reynold's Syst. of Med. 1871. Vol. III.
Fowler and Godlee	Diseases of the Lungs. London,1899.
Fowler	"Syphilitic Disease of the Lungs." Allbutt's Syst. of Med. Vol. V. pp. 311 et seq.
Gairdner and Coats	Lectures to Practitioners. Glasgow, 1888.
Greenfield	Path. Soc. Trans. Vol. XXVIII. pp. 248 et seq.
Greenhow	Path. Soc. Trans. 1865 to 1869. Vols. XVII - XXI.
Heller	"Die Lungenerkrankungen bei angeborener Syphilis." Deutsch. Archiv f. klin. Med. 1888. Bd.XLII.
Hirt	Die Staubinhalationskrankheit, 1871.

Hochsinger	Wiener med. Blatt. 1894. Nos. 20,21.
Handfield Jones	Brit. and Foreign Med. Chirurg. Rev. 1884.
Juergensen	Ziemssen's Cyclop. of Pract. of Med. Vol. IX., art. Interstitial Pneumonia.
Kidd	Lancet 1890.
Lewin	"Die Inhalationstherapie." art. in the Krankheiten Respirations Organe. 1865.
Lewis and Balfour	Public Health and Preventive Medicine. p. 446. Edinburgh, 1902.
Loew	Archiv für Physiologie, 1901.
Mitulescu	La Determination du Coefficient Nutritif Cellulaire. Paris 1902.
Petit et Thezard	"Tuberculose et urologie." Rev. de la Tuberculose, June 1904.
Philip	Pulmonary Fibrosis. Encyc. Med. Vol. VII. pp. 140 et seq.
"	Pneumonokoniosis. Encyc. Med. Vol. VII. pp. 145 et seq.
Powell	Diseases of the Lungs and Pleura,1886.
Rokitansky	Collected Works. New Sydenham Soc.
Shattuck	"Cirrhosis of the Lungs." Boston Med. and Surgl. Jnl. 1882.
Smith	Clinical Studies of Disease in Children. 2nd Edition. London, 1892.
Smith, Pye	"Pulmonary Cirrhosis." Allbutt's Syst. of Med. Vol.V. pp.91 et seq.

Stevenson and Murphy	A Treatise on Hygiene and Public Health. Vol. II. pp. 364 et seq. London, 1893.
Stokes	Treatise on Diseases of Chest, 1837.
Sutton	"Fibroid Degeneration of the Lungs." Med. Chirurg. Trans. 1865.
"	An Introduction to the Use of the Stethoscope. Edinburgh, 1825.
Tracey, Roger	Hygiene of Occupations. New York, 1889.
Walsh	Practical Treatise on Diseases of the Lungs. London, 1860.
Willis	Lectures on Pathological Anatomy. 3rd Edition. pp. 352 et seq. 1889.
"	Path. Soc. Trans. Vol. VIII.
Zenker	Deutsch. Archiv. f. klin. Med. 1866. No. II and 1878 No. XXII.